THE JOURNAL

OF THE

OKLAHOMA STATE MEDICAL ASSOCIATION

VOLUME XXVI MUSKOGEE, OKLAHOMA, MARCH, 1933. NUMBER 3

CHRONIC INDIGESTION

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There is perhaps no group of symptoms regarding which there is more misapprehension among physicians than symptoms arising from the stomach or felt in the region of the stomach.

There is no distinct disease entity known as "chronic indigestion." The syndrome of true gastritis occurs so rarely that it may safely be forgotten by all save specialists in gastro-intestinal diseases.

If one learned by simple statements rather than by an elaboration of those statements, this article would be complete in the single sentence: "There is no such thing as chronic indigestion." As this is not true and as there is a crying need for the early recognition, the accurate diagnosis, and sane, logical treatment of those conditions long neglected and belittled by the application of the misnomer "chronic indigestion," we are submitting this paper in the hopes that it may, in its small way, tear aside this shrouding term and awaken the practitioner to the underlying pathology, often of serious nature.

It is not that we expect to make any epical disclosures or revolutionize the study of medicine by new discoveries. We wish only to recall to mind, in a timely review, that which every physician knows or has known; so that by repetition we may keep before our minds the fact that vague gastro-intestinal symptoms are worthy antagonists and deserving, in every case, of exhaustive measures of diagnosis.

"Indigestion" is a much abused term commonly used to cover all forms of stomach disease. Strictly it means the non-digestion of food. This is a rare event among those who are not seriously ill. That digestion may fail in the stomach or in some part of the alimentary canal or that some parts of the food may escape digestion is common enough, but the human organism is provided with compensating mechanisms, so that if one organ in the digestive system fails to perform its duty, another is usually capable of taking its place. Digestive ferments are not often lacking, and there is rarely a rational indication for prescribing artificial ferments to supply a lack in the normal action of these organs, except dilute hydrochloric acid.

Formerly it was a favorite custom to explain the belching of gas from the stomach and the flatulent distention of that organ, as also the "sour" stomach, by saying these symptoms arose from the fermentation of food. Such an explanation gave rise to attempts to suppress fermentation by giving a host of intestinal antiseptics of varying degree of germicidal power. Fermentation does rarely occur in the stomach, however, ordinarily the formation of gas is slow and quite insufficient to account for the belching that many patients experience. In the majority of cases the gas present in the stomach consists of swallowed air. As a rule, even in cases in which much distress is produced by flatulence or belching, there is no fermentation in the stomach. The acid present in the stomach contents is seldom the results of fermentation but is produced by over-secretion of the gastric juice. It is essential, therefore, to ascertain the true origin of these symptoms before attempting to prevent them by the administration of injurious antiseptics.

However, the evil is usually not in the injuriousness of the medication so much as that treatment has been offered more or less empirically with no more than a cursory attempt at diagnosis. Once a patient is labeled dyspeptic he is disposed of with prescriptions for alkalies or digestants and promptly forgotten.

Diagnosis on the basis of the patient's

recital of symptoms without physical examination or the analysis of gastric contents or X-ray series is much too common. It may be said at the outset that there exists scarcely a symptom that is characteristic of any definite stomach disease. This may explain the readiness with which practitioners resort to such terms as "indigestion," "dyspepsia," "catarrh of the stomach," or the equally indefinite term, "stomach trouble" as a disgnosis.

While the existence of serious organic disease should never be overlooked, it is well to understand that only a small proportion of patients who come to the physician complaining of the stomach or of digestive disturbances have ulcer or cancer. The physician should not make or suggest a diagnosis of serious disease until he has proved its existence by appropriate physical and laboratory examinations, reserving them as potential possibilities until proven otherwise.

It should always be borne in mind that the stomach has important nervous connections with other organs by which it reflects, like a mirror, events taking place in other parts of the digestive system. Symptoms arising in the stomach may, in reality, depend on disease of the liver, gallbladder, appendix or large bowel. Neighboring organs not connected with the process of digestion or even remote organs may produce a reflex disturbance in the stomach.

An attempt to group together in a comprehensive classification, all conditions which may secondarily give rise to gastro-intestinal symptoms would lose its clarity and usefulness defeating its purpose through its very volume. While the classification presented can in no way be considered complete; it is a list of the conditions most usually encountered and which we entertain in mind while examining each patient with gastro-intestinal symptoms, ruling out or verifying each syndrome complex until a definite diagnosis is reached during the course of the examination:

I. Those intra-abdominal lesions most commonly giving rise to "dyspeptic" symptoms:

1. Cholecystitis.
2. Peptic Ulcer.
3. Gastro-Intestinal Malignancies.
4. Colitis.
5. Appendicitis. (Chronic).

II. Those extra-abdominal conditions prone to give rise to "dyspeptic" symptoms and usually requiring a gastric analysis to confirm or strengthen the diagnosis.

1. Pernicious anemia.
2. Pellagra.
3. Sprue.
4. Combined systemic disease.

III. Those unrelated conditions with "dyspeptic" symptoms as occasional secondary manifestations.

1. Acute infections.
2. Chronic infections, especially tuberculosis.
3. Circulatory and renal diseases.
4. Metabolic diseases.
5. Diseases of the genital organs.
6. Diseases of the C. N. S., functional and organic.

The intestinal symptoms of the last two groups, especially of the latter are but secondary minor manifestations and the diagnosis is usually apparent. Those in the first group call for careful differentiation.

Each of the conditions listed may give the identically same vague symptoms of gas, eructation, and belching, slight nausea and even vomiting. Pain may vary in its distribution. It may vary in its severity or in its interpretation by the patient from a sense of epigastric fullness or oppression, to intense cramp-like seizures or unbearable boring dull pain. An hysterical patient may make mountainous complaints of slightly exaggerated peristaltic movements while a plethoric minded individual with peptic ulcer and undoubted pain make but slight and passing comment of abdominal discomfort.

The relation of pain to food and hunger is of extreme importance and may be beautifully typical in peptic ulcer but can, too frequently, be confusing or lacking in definite ulcer or present in unrelated conditions.

Relief of pain by the ingestion of soda would indicate the presence of a condition associated with hyper-acidity and thoughts turn first to ulcer. However, one of our patients, and this is by no means unusual, had been taking soda by the advice of a physician for the past twenty years with varying degrees of sense of relief. Gastric analysis showed a complete lack of hydrochloric acid in both the fasting contents

and after a test meal. For twenty years physicians had been attempting to neutralize an acidity which did not exist. Twenty of the most valuable years of a patient's life were spent floating from physician to physician and from alkali to alkali. For twenty years the full realization of the patient's economic possibilities were more or less hindered while ten minutes work with the stomach pump and tube would have shown us that at least our medication was contraindicated.

We have herein presented the twenty-eight cases, in the order in which they presented themselves for examination, during the period from November, 1931, to May, 1932.

Hosp. No.	Sex	Age	Tetra Iodide G. B.	Gastric Analysis	X-ray Findings	History	Final Clin. Diag.
S. S. B. 4230	F.	62	Normal	Normal to high	Spastic Colon	Gas, Eructation, Burning lower abdominal pain.	Spastic Colitis
W. J. D. 4184	F.	—	Normal	Achlorhydria	Spastic Colon	Flatulence, pain of distention.	Spastic Colitis
R. H. C. 4192	M.	—	Normal	Normal	Atonic Colon	Constipation	Atonic Constipation
C. D. D. 4132	F.	42	Normal	Hyperacidity	Doudenal Ulcer	Typical Ulcer History.	Peptic Ulcer
P. M. S. 4212-4213	F.	40	Pathological	Hypoacidity	Pathological G. B.	Repeated attacks R. U. Q. pain. Jaundice fever for one week.	Operation — Pericholecystic abscess obstructing cystic duct and common bile duct.
S. J. W. 4215	M.	39	Normal	Hypoacidity	Indeterminate Rapid Stomach emptying time.	Three months distention without pain, nausea, vomiting relief by defecation.	Hypochlorhydria Gastrica.
J. M. M. 4040-4058	F.	43	Normal	Hyperacidity	Spastic Colon	Mucous stools thirty years. Typical ulcer with much gas formation.	Spastic Colitis
W. R. S. 4174-4175	M.	—	Normal	Normal	Colon spastic. Appendix segmented.	Typical ulcer history	Spastic Colitis Chronic Appendicitis.
W. H. J. 4199-4200	F.	49	Normal	Hypoacidity	G. I. negative. Liver shadow grossly enlarged.	Nine weeks weakness, pain and nausea.	Ca. Liver
T. G. 4118-4119	M.	38	Normal	Hyperacidity	Colonic Stasis. Rapid Stomach emptying.	Two months sharp epigastric pain. Dietary indiscretion.	Hyperacidity
E. H. 4231	F.	28	Normal	Normal	Delayed stomach emptying.	Burning epigastric pain. Recurrent attacks acute appen.	Chi. Appenedicitis
R. L. C. 4160	M.	38	Normal	Hyperacidity	Indeterminate	Epigastric discomfort.	Hyperacidity
P. Y. 4168	F.	—	Normal	Hyperacidity	Rapid Stomach emptying.		Hyperchlorhydria
E. P. 4209-4210	M.	43	Pathological	Hypoacidity	G. I. negative. G. B. pathological.	Acute R. U. Q. pain with shock jaundice.	Chronic cholecystitis
E. D. P. 4219-4225	M.	37	Normal	Normal	Doudenal scar with deformity of cap	Typical of Ulcer.	Duodenal Ulcer
L. C. T. 4166	M.	—	Normal	Normal	Negative	Negative	Chronic Appendicitis
H. B. 4148	M.	48	———	Normal	Colon-filling defect.	Severe pain and L. L. Q. tenderness.	Ulcerative Colitis.
W. H. D. 4143-4144	M.	62	Pathological	Achlorhydria	G. I. negative.	Seven weeks "flu" progressive jaundice without pain. Gas, distention.	Ca. head of pancreas
H. C. G. 4129	M.	39	———	Normal	Rapid stomach emptying.	Typical Chronic Ulcer history for years.	Chronic appendicitis
J. C. C. 4044	F.	—	Normal	Hyperacidity	Ulcer lesser curvature colonic delay.	Typical Ulcer history 1 to 2 years.	Gastric Ulcer
W. T. W. 4045-4046	M.	66	Pathological	Hyperacidity	G. B. pathological. G. I. series inadequate.	Failing health 10 yrs. Cramping pain three weeks. Blood in his stools.	Gastric Ca.

Hosp. No.	Sex	Age	Tetra Iodide G. B.	Gastric Analysis	X-ray Findings	History	Final Clin. Diag.
H. T. G. 3997	M.	32	Normal	Normal	Ulcerous Area pylorus.	Two weeks typical ulcer, history of much liquor.	Gastric Ulcer (Alcoholic).
F. N. K. 3851	M.	40	Normal	Hyper-acidity	Ulcer of stomach.	Twelve years seasonal ulcer history with hematemesis.	Gastric Ulcer
H. W. B. 3859	M.	66	Normal	Achlor-hydria	Filling defect of stomach	Three months signs of obstruction with epigastric mass.	Gastric Ca.
D. A. P. 4043	F.	28	Normal	Hypo-acidity	Colonic Stasis.	Six weeks R. U. Q. pain referred to scapula hunger pain.	Hypochlorhydria Functional Constipation.
K. P. 3875	M.	37	Normal	Hypo-acidity	Gastroenterostomy.	Twelve years ulcer history.	Duodenal Ulcer
R. R. T. 4014-4016	F.	69	Normal	———	Colonic Stasis Ileocecal incompetence.	Gas R. L. Q. pain several years.	Chronic appen.
J. J. D. 4076	M.	40	Normal	Hyper-acidity			Hyperchlorhydria

The following general statistics gleaned from this study may be of passing interest:

Women are most usually thought of as the "complaining" sex and are more able to spare time in which to be sick. However, only 39.3% of our patients were females and 60.7% males.

In the twenty-one who showed no reticence as to age; the average age was forty-five years with no patient younger than twenty-eight or older than sixty-nine presenting themselves for examination.

This group does not include or take into consideration those cases of typical acute and chronic appendicitis admitted and operated after a mere history and physical. Many of these, however, gave a long past history of gastro-intestinal disturbances of slight nature.

Many of these cases were suffering from more than one condition so that our total listed will be greater than 100%. Six cases or 21.4% showed a colitis, three of which suffered from marked constipation. Six cases or 21.4% gave X-ray evidence of peptic ulcer which was borne out by the clinical findings. Three cases or 10.7% were cases of cholecystitis. Five cases or 17.8% had chronic appendices. Four cases or 14.2% showed hyperchlorhydria without demonstrable cause.

Fully 50% of these diversified conditions had been indiscriminately treated with soda.

It can be seen also that the symptoms listed as "typical of ulcer" are symptoms, not of the ulcer *per se*, but of the disturbed gastric acidity.

In these cases we have not considered hyperchlorhydria and hypochlorhydria as a distinct disease entity; but rather as a demonstrable symptom of some under-lying disease complex. In those cases in which we have been unable to attribute the secretory disorder to any organic lesion, and in which we have been unable to locate any pelvic, toxic or extra-abdominal lesion, of which it may be a reflex manifestation, we have hesitantly labeled as of psychogenic origin and purely functional.

The symptoms presented are due to the disturbed secretion. These are combatted with diet, alkalies or dilute hydrochloric acid as the case demands and the patient's symptoms relieved. The patient, however, is not dismissed from mind, but is watched for the appearance of any condition of which the secretory disturbance may have been a forerunner.

It may be well to recall that hyperacidity and especially hypoacidity show a familial tendency and may long be present without symptoms. This is particularly true of families in which pernicious anemia has appeared. Hypochlorhydria may have been noted long years before the manifestation of anemia; and, as is often the case other members of the family may show a hypochlorhydria with or without apparent symptoms.

The ideal routine of examination is that

of the methodical specialist and should contain the following:

1. Meticulous history.
2. Complete physical examination.
3. Gall bladder series after tetraiodide administration.
4. Gastric analysis of the fasting contents and after a test meal.
5. Complete G. I. series.

After such a procedure, little of importance can escape our attention and the percentage of our mistakes are lowered to a minimum. Economic conditions and other circumstances may influence us to delete certain items of the above routine as unnecessary or superfluous in the individual case. But after all, where so much confusion is possible, are we ever warranted to give less than the best we have?

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FUNDAMENTALS OF ELECTROCARDIOGRAPHIC INTERPRETATION

In a foreword to the first of a series of articles by J. Bailey Carter, Chicago (Journal A. M. A., Oct. 1, 1932), on the fundamentals of electrocardiography interpretation the editor of The Journal points out that electrocardiography has been gradually establishing a place for itself in clinical practice. Nevertheless, a large number of physicians have not been able to develop a working knowledge of the subject. For this reason The Journal presents a series of articles outlining the fundamentals necessary to understanding it. In the preparation of this series, various other contributions on the subject were consulted and the statements of authors rewritten, rearranged or left practically unchanged to meet the requirement of simplicity and clearness. This is not a report of research. Instead, Dr. Carter has avoided the points that are still debatable. In the preparation of the material, Drs. Arno B. Luckhardt, Samuel R. Slaymaker, N. S. Davis III and L. N. Katz, Mr. W. B. Andrews and Mr. H. J. Holmquest have aided with suggestions and advice. The electrocardiograms and case histories have been developed from the collections in the Central Free Dispensary, Passavant Memorial Hospital and the Cook County Infirmary, and are from the departments of Dr. Samuel R. Slaymaker, Dr. C. C. Maher and Miss Myrtle Sands. It is realized that the vast majority of physicians are not in themselves possessed of the necessary apparatus for electrocardiographic studies. Nevertheless, such service is now available in many hospitals, clinics and laboratories. An outline of the presentation of this subject to be followed in this series of articles is given and, following an introduction to the general subject of electrocardiography the author discusses the following subheadings: (1) the value of the electrocardiogram in clinical practice; (2) physiologic basis of electrocardiography; (3) origin and action of the heart currents; (4) electrocardiographic technic; (5) the process of electrocardiography; (6) artefacts and their origin; (7) waves of the normal electrocardiogram, and (8) individual wave changes.

THE DIAGNOSIS AND TREATMENT OF THE MORE COMMON ACUTE ABDOMINAL CONDITIONS

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A great many lengthy papers have been written on the "acute surgical abdomen" in recent years. This emphasizes the importance of this subject and too much cannot be said about this common condition, met with so frequently in medical and surgical practice. The author has written somewhat in length on this subject, the article appearing in the State Journal of February, last year. In today's paper I have made no attempt to discuss the subject in a general way, but instead I propose to discuss some of the common acute abdominal conditions in the order of their frequency.

I shall take up first the subject of acute appendicitis. A study of this condition in a large series of cases shows some interesting facts:

1. As long known, it is a disease of adolescent and early adult life, 68% of the cases occurring between the ages of 10 and 30. A fact not always appreciated is that it occurs more frequently in children under 13 years than in any decade over 40.

2. A study of the time interval between the onset of symptoms and hospital admission averages between 3 and 4 days. This long interval between the onset and admission is due either to the failure of the patient to call a physician, or to the failure of the physician to make a diagnosis, or to the refusal of the patient to submit promptly to surgical treatment after diagnosis has been made, or, finally, to the desire of the physician to treat the condition medically.

3. Further study reveals that 27% have had previous attacks, 72% being hospitalized in their first attack. When statistics reveal that almost 40% of the cases of acute appendicitis have already perforated on admission to the hospital, we realize the danger of treating expectantly either the first or subsequent attacks in the hope that they will subside.

4. Considering the mortality rate in chronic cases and acute unperforated cases it is not more than 1%. In cases with perforation and abscess formation, it is between 10 and 15%, and cases with

spreading or general peritonitis have a mortality rate around 50%. It is to be emphasized that it is the early diagnosis of acute appendicitis with which we are concerned and not the diagnosis after abscess or peritonitis has developed.

Acute appendicitis is the most common acute abdominal condition which we see, and its very frequency warrants its serious consideration in the diagnosis of any acute abdominal condition. General abdominal pain in the early period of the attack occurs in 75% of the cases, but localized pain and tenderness in the appendiceal region occurs in 95%of the cases—pain being the most important single symptom of the disease. Nausea and vomiting are the next most constant symptoms occurring in 75% of the cases. It is to be emphasized that vomiting usually is not frequent; it consists of one or two attacks coming on soon after the onset and then ceasing until peritoneal irritation develops. Localized tenderness and muscle rigidity are dependent upon the relation of the inflamed appendix to the parietal peritoneum. They are usually present, but if the appendix is retrocaecal or deeply placed in the pelvis they may be absent. In such cases deep palpation may reveal the local tenderness and rectal examination may be of aid.

In children it is much more difficult. Neither the symptoms nor signs are so constant. The fever and leucocytosis are as a rule higher and the difficulties in elicting localized tenderness are greater. In the absence of a diarrhea (important) an attack of abdominal pain with nausea, vomiting, fever and leucocytosis should be considered acute appendicitis until proven otherwise.

The errors in the early treatment of acute appendicitis are chiefly two. Most important, I think, is the attitude of the physician towards the disease. We find there is still a tendency to treat the disease expectantly in the hope that the attack will subside. Undoubtedly, many attacks do spontaneously subside; but it cannot be foretold that any given attack will subside. The dangers of good surgery as before stated is 1% or less, depending on the stage of inflammation.

Another error in the early treatment of the disease is due either to the failure of the physician to make the diagnosis or his lack of understanding of the so-called medical treatment of acute appendicitis.

The fundamental principle of this form of treatment, as you know, is rest or quietness of the G. I. tract. We know from clinical and experimental observation that an inflammation of the tissues or organs spreads less rapidly and is more likely to subside if the involved tissue or organ is at rest or quiet. For the intestinal tract rest is secured by withholding foods and fluids by mouth, by the administration of morphine and by withholding all purgatives. Not only is this of help in limiting the spread of inflammation, but it is also of great help in the formation of adhesions about the appendix so that if perforation does occur there will form a localized abscess rather than a general spreading peritoneal infection. It is a matter of record that one-third of the cases of acute appendicitis received an initial purge. Now the physician is not always responsible for this error, for it is common knowledge that patients will take purgatives without consulting or even against the advice of the physician. But, nevertheless, the administration of cathartics by physicians in acute appendicitis is not unheard of.

Acute Cholecystitis is next in frequency to acute appendicitis. It is a condition which may be unassociated with stone, but usually is associated with stones in the gall bladder, one or more of which temporarily or permanently occludes the cystic duct. When the symptoms and signs are typical, diagnosis is easy; but when atypical one must rely on experience and often we may help ourselves by the use of the X-ray. Gangrene and perforation occur more commonly in the old than in the young. The mortality rate for operation is no higher in the acute stage of cholecystitis—providing gangrene and perforation have not occurred—than when performed during a quiet interval. Therefore, in my opinion, it is best to operate these cases early because the mortality is high after gangrene and perforation have occurred.

Acute Perforation of Gastric Duodenal Ulcer. This condition is next in frequency to acute cholecystitis. A review of the histories of these cases shows that in about 80% there were symptoms dating from a period of three months to twenty years before perforation. In the remaining 20%, history was indefinite or no history of previous ulcer symptoms existed.

The picture of the acute perforation of gastric or duodenal ulcer is a sudden, intense pain in the epigastrium, followed in

half the cases by nausea or vomiting. If seen very early, there may be pallor and other symptoms of shock, or shock may be absent. The abdomen is rigid, more so than usually is seen in other acute abdominal conditions and it may even be retracted. There is abdominal tenderness more marked in the upper half of the abdomen. The temperature is normal or subnormal. The pulse is moderately accelerated and the leucocytis increased. Later, with spreading peritoneal infection, the abdomen distends, leucocyte count drops and pulse accelerates. The diagnosis is usually made in 80% of the cases. The most common error is in confusing perforated ulcer with acute appendicitis, next acute cholecystitis, acute pancreatitis. lead colic and small strangulated ventral hernia in order of frequency.

The importance of early diagnosis and prompt surgical treatment in no other condition is better illustrated by a study of mortality rates considering the time interval between perforation and operation. In cases operated upon within the first twelve hours the mortality rate is about 20%, while in those cases operated between twelve and twenty-four hours after perforation the mortality rate climbs as high as 70%

Acute Intestinal Obstruction. This condition occurs fourth in frequency in the diagnosis of acute surgical abdomens. As we see this condition clinically we may conveniently classify the patients who present its symptoms into two groups:

1. Those who are admitted to the hospital primarily for this condition.

2. Those who develop the condition in the hospital following operation for some other surgical disease.

Acute intestinal obstruction in the first group follows a variety of causes, such as adhesions or bands, the result of previous operations, strangulated hernias, intussusception, volvulus, tumors of the intestine enterolith, tuberculous peritonitis, Meckel's diverticulum and internal strangulated hernia in order of frequency.

High intestinal obstruction is one of the varieties in which errors in diagnosis are prone to occur. The symptoms are intense cramp-like pain, vomiting, a normal or a subnormal temperature, accelerated pulse and leucocytosis. The abdomen may fail to present even a slight distention and palpation may be negative. Efforts to evacuate the bowels will in all probability be successful for twenty-four or forty-eight hours because the enemas empty the bowel below the high obstruction, so don't let this mislead you. The confusing elements here are the absence of abdominal distention and the pseudo bowel movement. A great aid in the diagnosis of these high type cases is fluoroscopy of the abdomen giving barium by mouth. The barium will be seen passing through the pylorus and stopping at the obstructive point in the duodenum or upper jejunum, the point of obstruction.

It is evident that the mortality in this disease is dependent on the time element between onset of symptoms and operation, just as it is in acute perforation.

About the second group of cases, those that develop symptoms of obstruction in the hospital following surgical procedures.

The obstruction may be mechanical and incomplete or complete, or it may be due to paralytic ileus.

As before stated the author made no attempt to discuss all of the conditions which might produce an acute surgical abdomen, but instead confined himself to the four most common causes met with in the order of their frequency. In closing allow me to quote the closing paragraph in a prior paper of mine on this same subject.

Acute abdominal conditions as a rule are always urgent and there must be little delay in these cases. I think one should never wait longer than a few hours. The patient should be placed where immediate operation can be done if necessary. In the few cases where a positive diagnosis has not been thoroughly established and the patient's condition fails to improve, exploration is indicated in the hope that the condition will be discovered and treatment instituted. We know that early operation often saves a life when in all probability delay would prove fatal.

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DRIED LACTIC ACID MILKS AS A LONG CONTINUED DIET FOR INFANTS

Because of their inability to find references in the literature regarding the subject, Julius H. Hess, I. McKy Chamberlain and Louis S. Robins, Chicago (Journal A. M. A., April 9, 1932), started a comparative feeding study with the hope of evaluating the effect on growth and development of infants following long continued feeding with dried lactic acid milks. The clinical results indicate that dried lactic acid milks can be used under the same conditions as cultured sweet milk and cow's milk plus U. S. P. lactic acid.

X-RAY THERAPY IN GRAVES DISEASE

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Experience has taught us that the X-ray is of no great value in the treatment of colloid and adenomatous thyroids although a few of the latter have been exposed to the rays and apparently benefitted. The exophthalmic syndrome however is definitely radio-sensitive and for this reason a short discussion of the disease is in order.

For one of the best definitions of exophthalmic goiter we are indebted to Dr. Israel Bram of Philadelphia. He states that this condition is a chronic (rarely acute) neuro-endocrine dysfunction, characterized by an increased metabolism, excessive appetite, loss of weight, emotionalism, tremor, persistent afebrile heart-hurry, weakness, dermographism and usually, though not constantly, a hyperplasia of the thyroid and exophthalmos.

In regard to diagnosis, I am reminded of Dr. A. E. Hertzler's article, "A Country Doctor Takes A Look At Stomach Complaints," in which he states that a physician should be able to make ninety per cent of his diagnoses in his B. V. D's. I feel that any doctor here tonight armed with Dr. Bram's definition and clothed in the manner just described could make a diagnosis of the type of goiter in question. In borderline cases however we can always make use of the basal reading, the Goetsch test, cinchona tolerance of Bram and voluntary apnea test of Bartlett.

The etiology of Graves disease is obscure but in general we may say that the condition occurs in both sexes, at all ages, and in almost every clime. Heredity seems to play a part in about 10% of the cases.

Graves disease is seen most often during periods of greatest sexual or adult activity. For every male case one sees about four females. Accounts of worry and emotional upsets are common in the histories of many cases of Graves' disease.

Another striking fact is that many of these patients date the onset of their nervousness and emotional instability from a preceding infection.

There are numerous theories regarding the pathological physiology of this disorder but to me the neuro-endocrine dysfunction and imbalance of the vegetative nervous system as set forth by Bram, Sajou, et. al., is the most plausible. The primary cause of this dysfunction and imbalance is at present unknown but most investigators feel that infectious foci, if not the exciting cause, at least aggravate the syndrome by their presence.

The gland in Graves disease presents the pathological picture of hyperplasia. Macroscopically the organ is usually increased in size but this finding need not be present in many typical cases. In other words there is no etiological relationship between the size of the gland and the severity of the syndrome. The enlargement when present is usually symmetrical. The surface is smooth, but a cut section presents a homogeneous structure not seen in ordinary parenchymatous goiter, with little or no visible colloid. There is an increase in the number and size of blood vessels. Microscopically there is an infolding and crowding of the cells upon the acinar spaces; an increase in the number and size of the cells; an increase in the number of alveoli and a diminution of alveolar colloid and its contained iodine (Bram).

In the majority of instances there is a persistently enlarged thymus gland. Kocher found enlarged thymus in fifty per cent of his cases; Blackford in a study of 74 autopsies, found that in all subjects under forty years there was a persistent thymus. Outstanding authorities now irradiate the thymus in all cases of Graves disease This is of great moment in post-operative cases suffering a recurrence of symptoms. Bircher in 1923, by transplanting particles of the thymus gland of patients suffering from Basedow's disease, was able to produce typical Basedow's disease in dogs.

Before enlarging on the subject of X-ray therapy I wish to quote Bram to the effect that, "Graves disease is not a pure hyperthyroidism, but a widespread affection, the predisposition to which is probably congenital." He states further that, "the rationale of both surgery and roentgenotherapy depends upon the theory that Graves disease is a pure hyperthyroidism." Thus the surgeon cuts out the supposed offender while the roentgenologist brings about the same result by the more subtle method of bombarding the gland with X-rays of extremely short wave length. Either method or a combination of both is considered good therapy at the present time. We must admit with Bram, however, that both methods are

only constituents of a broad regime of non-operative therapeusis.

Our method of applying the X-ray consists of crossfiring the gland through two to seven different skin areas, depending on the size of the organ and the severity of the symptoms. This series is usually administered at one sitting and is repeated every ten days until the basal reading drops to and remains in the neighborhood of +15. For the information of any radiologists present the exact set up is as follows: 5 milliamperes, 135 kilovolts or about a 9 inch gap, 10 minutes exposure time over each skin area with a 6 millimeter aluminum filter and a target skin distance of 10 inches. The energy is developed by an oil-immersed transformer with a rotary rectifier and is controlled by an autotransformer unit. In blonde individuals one thickness of sole leather is used in addition to the 6 millimeters of aluminum.

Each skin area is carefully marked with an indelible pencil and leaded off. The size of the skin area is varied to suit the case but ranges between three-fourths of an inch to two inches. The smaller the skin area the more opportunity for crossfiring and as a result the greater the depth dose. Small skin areas which are close together are heavily shielded with quarter inch lead and an area about one foot square is protected with lead one-sixteenth of an inch thick. The body from the neck down and any visible portion of the scalp is covered with lead rubber. We feel that this amount of protection is very essential when the operator is using heavy cross-fire on such a small area as the thyroid. In our opinion the operator should not put too much faith in the ordinary lead glass bowl in thyroid therapy.

As a protective measure against telangiectasis from two exposures inadvertently given over one skin area an indelible dot is placed in the center of the exposed area just before throwing the X-ray switch.

The time of exposure is controlled by two interval timers and the observers watch.

The thyroid in Graves disease is especially sensitive to heavy cross-firing with the above or similar technique. The rays probably destroy the gland by an atrophic action on the cells *per se* and by producing a swelling of the T. intima of the capillaries and terminal arterioles. The capillaries and terminal arterioles finally become obliterated and portions of the gland degenerate from lack of blood supply. Some observers have also noticed a round cell infiltration and later a moderate degree of fibrosis. Secretion of the gland is diminished early probably because the secretory cells are first affected. Next there is a gradual atrophy of the gland with a decrease in size of the neck circumference.

A decrease in nervous symptoms is usually the first clinical evidence of improvement and may be observed as early as three weeks or as late as two months, depending apparently on the vascularity of the gland. Large pulsating growths with a shrill bruit on auscultation are especially slow to respond. As the nervous manifestations improve the patient begins to show some gain in weight. The tachycardia as a rule diminishes very slowly. Glandular enlargement and exophthalmos are usually the last symptoms to leave and the latter is often permanent.

An X-ray "cure" often requires six to eighteen months but the patients are usually able to work all through the course of treatment. In fulminating cases rest in bed between exposures is a valuable aid in the therapeutic regime.

In regard to the choice of treatment we feel that each individual case may possibly, but not always, require the services of an internist a roentgenologist and a surgeon, in the order named, before permanent remission takes place. Bartlett in Radiology, March, 1932, speaks of permanent stabilization of these patients rather than cure and urges closer cooperation between the internist, radiologist and surgeon in an effort to bring about this permanent stabilization.

Statistics seem to show that surgery alone and radiology alone produce about the same percentage of the so-called cures. For this reason it seems logical that radiology should always precede surgical intervention. It should be used instead of superior pole ligations and should be used long enough to show either failure or success. Success is usually the rule. Williams reported 200 carefully controlled cases in Radiology for March, 1932, with 80.5 per cent cures. Twenty-seven cases or 13.5 per cent were improved, making a total of 94 per cent either cured or improved. Eight cases or 4 per cent recurred after one year. Five of these were re-treated and cured. Only two cases developed sub-

thyroidism and only one had telangiectases.

Surgery at times produces occasional patients with a combination of subthyroidism and Graves disease super-imposed and the same is true of X-ray, but to a lesser degree. Dr. Bram, in Archives of Physical Therapy for September, 1932, reported a case of thyroidless Graves disease from X-ray exposures.

According to Walter, Anson and Ivy, Radiology, January, 1931, the parathyroids are apparently immune to therapeutic X-radiation. Pfahler in Radiology, May, 1932, states that none of his cases have ever developed tetany after X-ray therapy.

Menville in Radiology for March, 1932, quotes Soiland, Costolow and Meland to the effect that reports from prominent surgeons and pathologists have discredited the belief that irradiated thyroids are more difficult to operate. They say that difficult operation is often met with in cases in which there has been previous thyroiditis with fibrosis and that it is impossible for the surgeon and pathologist to pick out cases which have had previous irradiation.

REPORT OF CASE

Mrs. W. M. L., age 43, first came to the office November 9, 1930. History was irrelevant except for the fact that she had recently suffered an attack of "flu." She stated that in spite of an excellent appetite she had been losing weight and was very weak, especially in the knees, when climbing stairs. Her heart bothered her by beating too fast on the slightest exertion and she complained of swelling of the legs below the knees. She also complained of increasing nervousness and irritability and the passage of large quantities of urine. (Urinalysis negative to routine examination). Physical examination revealed an apparently undernourished woman of about stated age. She was very nervous and jumped at the slightest sound or unexpected movement on the part of the observer and was continually moving some part of her body. The eyes gave the impression of great fear. Pupils were widely dilated but reacted to light and accommodation. The sclera was visible between the upper lid and pupil. The neck presented a very pulsatile enlargement of both lobes and isthmus of the thyroid, the left lobe being visibly the largest. There was a bruit on auscultation. Observation of the hands revealed a marked tremor. Heart rate 120 per minute, pulse synchronous, temperature 98 degrees, respiration 20. Auscultation revealed no shocks or murmurs. Blood pressure 150-60, pulse pressure 90.

The patient reported for a basal metabolic reading November 11, 1930. Her weight was 120 pounds without clothes, pulse 110 per minute and respiration 20. Her basal reading was plus 57. From December 5, 1930, to March 16, 1931, she received seven series of exposures of the technique described above and on March 16, 1931, her weight was 138 pounds and pulse was 100. Patient was so much improved she stopped treatment of her own volition and stated she did not even want a basal metabolic reading.

Following this the patient left town and did not return until January 4, 1932. She complained of all her old troubles. Her weight however was 137 pounds, pulse 100, and the pulsating tumor was no longer visible in the neck. Her basal reading was plus 45. From January 4,1932, to April 16, 1932, she was given ten series of exposures at intervals of ten to fourteen days. Basal reading March 19, 1932, was plus 42. On April 16, 1932, we decided patient was stabilized and advised another basal reading which the patient refused. Patient was not seen again until October 21, 1932, at which time she stated she was feeling fine. Her weight was 135, pulse 85, and blood pressure 120-80. We would like to call attention to the pulse pressure which at instigation of treatment was 90 and now is 40. Patient submitted to basal reading, November 2, 1932, which was plus 16. Weight was 142 pounds, pulse 92 and respirations 16.

On the basis of case reports in the literature and our own experience with this one case we are inclined to summarize with Holzknecht: "Only those patients suffering with Basedow's disease should be operated on, who do not respond to irradiation, cases with large struma, compression of the trachea, or very acute cases of Basedow's disease which do not respond favorably to irradiation."

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EFFECT OF KNEE-CHEST POSITION AND POSTURAL EXERCISES ON POST-PARTUM RETROVERSION

Goodrich C. Schauffler, Portland, Ore. (Journal A. M. A., Aug. 27, 1932), put alternate patients (odd numbers) in a group of 169 deliveries on knee-chest position and postural exercises, whereas alternate patients (even numbers) were not given such measures. Examination after six weeks revealed a substantially higher incidence of postpartum retroversions in the group subjected to the so-called corrective measures. The authors believe that it is fair at least to conclude that the use of such exercises does not justify the confidence that has been placed in them. It seems wise even to seek for factors in the use of such exercises that may actually retard the tendency toward involution and return to the normal anterior position.

FORCED SPINAL DRAINAGE IN ITS RELATION TO INFECTIONS OF THE CENTRAL NERVOUS SYSTEM

George M. Retan, Syracuse, N. Y., (Journal A. M. A., Sept 3, 1932), used forced spinal drainage in twenty-one cases of various infections of the central nervous system. In four autopsies in cases of septic meningitis no hydrosis of any organ was found. There was evidence of a washing of fluid through inflammatory areas. Products of inflammation were washed from the depths of affected areas. The field of greatest usefulness for forced spinal drainage was in diseases of the central nervous system characterized by perivascular round cell infiltrations. Forced spinal drainage was shown to be a safe procedure. Over two thousand hours of treatment have produced no alarming symptoms.

STREPTOTRICHOSIS

From their review of the literature and the observation of a case of streptotrichosis, Maurice Kovnat and Cornelius Mezei, New York (Journal A. M. A., Dec. 10, 1932), conclude that streptotrichosis, in both acute and chronic forms is probably more common than is generally believed. The etiologic factor is a branching mold, very pleomorphic botanically, but showing a typical pathologic entity. The chromogenic granules are so pathognomonic that their presence alone—easily detectable macroscopically—determines the diagnosis of this disease. The case reported by them shows the typical, clinical course of acute pulmonary form of the disease which followed on the ulceration and removal of a chronic skin nodule of the same etiology. The possibility of a relatively innocuous skin lesion harboring virulent micro-organisms should not be overlooked.

USE OF IODIZED OIL IN DIAGNOSIS OF CHRONIC MAXILLARY SINUSITIS*

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The use of iodized oil as an aid in diagnosis of chronic maxillary sinusitis has been more or less used since 1926. During that year Fraser[1], Goodyear[2], Proetz[3], and MacCready[4], reported a series of cases with their observations after injection of lipiodol into the nasal accessory sinuses. Many papers have been published within the past few years by both rhinologists and roentgenologists advocating its use.

I do not believe this question has been definitely settled as yet, although in the majority of cases of chronic maxillary sinus involvement, the injection of opaque oil will usually show a filling defect, if antrum is diseased, which shows up on operation to be a thickened diseased membrane.

The diagnosis of chronic maxillary sinusitis is difficult, an ordinary roentgenogram of the sinuses usually show very little, cloudiness of one or both sides may be due to position of patient's head or a thickened bony wall of the antrum. Transillumination of the sinuses is not reliable although a valuable aid. Irrigation of the antrum may or may not show pus.

Given a case with evidence of focal infection, whose complaint is headaches, vague pains in face, or bronchial cough, the ordinary roentgenogram of the sinuses is not enough, the picture may show some changes, but as a rule will leave us in doubt. Filling of the antrums which requires very little additional effort and is practically painless will clear up the diagnosis in the majority of the cases. Simon[5] rules antroscopy superior to other methods of diagnosis of maxillary sinusitis. I have had no experience with this method, although I believe this is another important step in diagnosing this condition.

He says. "One must become accustomed to recognizing the changes in the mucous lining of the sinus, regardless of how well he knows the anatomy of the nose and the paranasal sinuses, this ability to recognize the changes will be acquired only by train-

*Read before the Section on Eye, Ear, Nose and Throat, Oklahoma State Medical Association, May 25, 1932, Tulsa, Oklahoma.

ing and experience in seeing sinus mucosa."

Osmond[4] in a paper read before section on radiology in Philadelphia, June, 1931, says, "The rhinologists have received much assistance in the diagnosis of sinus disease since the roentgenologists have been making sinus examinations before and after the injection of iodized oil. Such roentgenograms detect thickening of the membrane lining the sinuses, and indicate the degree of thickness of the hyperplastic tissue, moreover, certain indentations and irregularities indicate the presence of polyps."

A complete history is essential in these cases, as Proetz has shown filling defects in a number of allergic patients after injection with iodized oil. Goodyear[7] has shown filling defect one week after an acute coryza.

The injection of oil should be delayed if either of these two conditions are present until after the height of the attack.

I have been making a practice of injecting oil in the maxillary antrums if there is a question of a chronic involvement, for the past year and a half, and I am convinced that it is one of the major points in the diagnosis. This procedure used in conjunction with other diagnostic aids at hand will save many operations on normal antrums.

According to Proetz[5] in the case of the normal sinus, the oil shadow conforms closely to the bone shadow. There should be a thin even line of light between them. The normal sinus mucosa varies in thickness from .5 mm. to 1 m.

CASE REPORTS

Case No. 1. White male, aged 35.

Present complaint: Post-nasal drip-recurrent head colds, recurrent attacks frontal headache, loss weight, and cough.

Transillumination. Right maxillary and frontal transilluminated well. Left, faint transillumination through maxillary, filling with iodized oil shows a marked filling defect on left side.

Right antrum retained 19 cc's oil—left 4 cc's oil. Left Caldwell-luc was done, with relief from symptoms. Left antrum was found to be filled with a large, thickened, cystic membrane.

Case No. 2. White male, aged 32. Referred for check-up sinuses.

Present Complaint: Pain in right arm and shoulder.

Normal filling—postero-anterior position — patient lying on face. Proetz advocates the partial filling and the horizontal beam. He has amply demonstrated no sinus can be filled above its normal ostium.

Tonsillectomy done five years ago. Examination revealed large tonsillar tags in both sides. No transillumination through either maxillary antrum. Frontals transilluminated well. Filling with iodized oil disclosed no filling defect. Right antrum retained 19 cc's, left 20 cc's oil. Removal of tonsillar tags brought about relief from his symptoms.

Case No. 3. White male, aged 41.

Present Complaint: Post-nasal drip (foul smelling) for past five years. Occasional headache. Sinuses transilluminated well. Filling with iodized oil shows no defect on right, left does not appear completely filled. Right antrum retained 20 cc's oil, left antrum retained 12 cc's oil. Patient refused operation. Instillation of .5 per cent ephedrin solution by displacement, after method of Proetz was resorted to, so far four irrigations have been given with some improvement. A filling defect is seen on floor of left antrum. An intranasal window resection with removal of membrane from sinus floor would be indicated in this type of case.

Case No. 4. White female, aged 27 years.

Present Compaint: Severe headache, frontal type. Post-nasal discharge. Feel-

ing of general weakness and loss of weight. Has noticed present symptoms for past four or five years. Had operations on her left antrum three years ago.

Transillumination. Right antrum transilluminated well. Left, transillumination fair, pus on irrigation left. Iodized oil was injected into both antrums. Right antrum retained 20 cc's oil, left 5 cc's oil. Left shows marked filling defect. Left Caldwell-luc was done. Antrum found to be filled with a thickened membrane and large amount of polyps.

Case No. 5. White male, aged 39 years.

Complaint: Recurrent head cold. Pain at times over right side of face. Post-nasal drip and recurrent headache. Transillumination. Both antrums transilluminate well. Iodized oil was injected into both maxillary antrums. Right antrum retained 13 cc's oil. Left antrum retained 19 cc's oil. Right antrum shows a filling defect confined to the floor.

Right antrum shows a filling defect confined to the floor.

An intra-nasal window resection was done on right antrum and a thickened membrane was removed from the sinus floor. Marked improvement was noted in his condition.

Case No. 6. White female aged 53 years.

Compalint: Pain over left side of face, post-nasal dripping and continuous head cold. Symptoms noticed for past two years. Both antrums were injected with iodized oil. Right antrum 21 cc's oil. Left antrum retained 12 cc's oil and shows a marked filling defect. A malignancy must be thought of in this case. Patient refused operation.

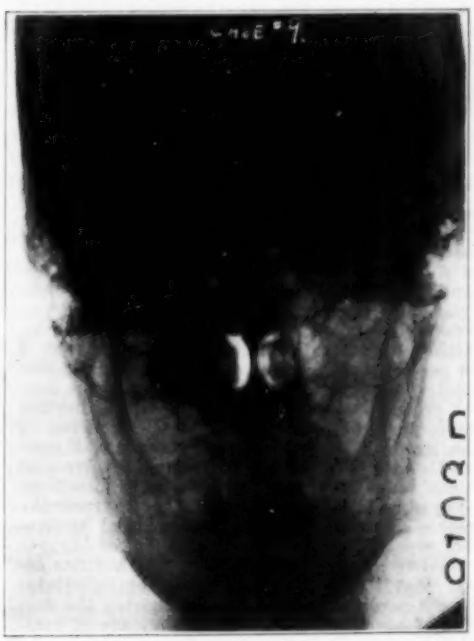
Case No. 7. Shows a normal filling, five days following an acute coryza. Antrum retained 23 cc's oil and shows no filling defect.

Case No. 8. White male, aged 31 years.

Compalint: Nasal obstruction, attacks sneezing, post-nasal dripping and occasional asthmatic attack. Examination showed both nostrils filled with polyps. Iodized oil was injected into both antrums. Right antrum retained 13 cc's oil. Left antrum retained 16 cc's oil. A ragged filling defect was found on both sides. Removal of nasal polyps and bilateral ethmoidectomy have been done. Irrigation by displacement method is now being carried out. Patient shows some improvement in his condition. Filling defect probably due to allergic condition.

Case No. 9. White male, aged 32 years.

Complaint: Periodic attacks of severe pain over left side of face and head accompanied by tearing of left eye, of four



No filling defect. Transillumination dark.

years duration. Advised on several occasions to have a radical operation done on left maxillary antrum. Transillumination dark. No pus on irrigation of left antrum. The left antrum was filled with iodized oil. Retained 19 cc's. Antrum appeared normal in outline, no filling defect.

This patient was referred to the internist who made a diagnosis of trigeminal neuralgia. An alcohol injection brought about relief from his symptoms for about six months. His symptoms reappeared, he was then referred to Dr. Frazier, Philadelphia, who sectioned the second division of the sensory portion of the nerve. He is now free from pain.

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TREATMENT OF RHEUMATIC FEVER PATIENTS WITH AND WITHOUT SALICYLATES: CLINICAL AND ELECTRO-CARDIOGRAPHIC STUDY

Thirty-three patients with rheumatic fever who received adequate salicylate therapy and thirty who served as controls were compared by A. M. Master and Alfred Romanoff, New York (Journal A. M. A., June 4, 1932), clinically and as to detailed electrocardiographic evidence of myocardial involvement. No essential difference was discovered. In the rheumatic fever patients, whether or not salicylates were administered, a 100 per cent involvement of the myocardium was found. The duration of hospital stay in the control group was forty-six days; in the salicylate group, forty-two days. One death occurred in each. group. Polycyclic course, occurrence of pericarditis with or without effusion, and involvement of the lungs and other viscera were practically the same in the two groups. The electrocardiographic changes indicative of myocardial involvement revealed a surprising coincidence of changes in the two groups, qualitatively. The authors believe that there is no evidence that salicylates prevent cardiac complications or shorten the duration of hospital stay, although they are at present the most efficient antipyretics and analgesics for the treatment of acute rheumatic fever.

PHRENICECTOMY AS AN AID IN THE TREATMENT OF PULMONARY TUBERCULOSIS

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Tuberculosis has existed from the most ancient times, Hippocrates having described very accurately the clinical manifestations of pulmonary tuberculosis. Since the time of Koch's discovery of the tubercle bacillus in 1882, scientific medicine has put forth every possible effort in an attempt to find a cure for this widespread disease which takes such a heavy toll each year. While general principles must guide us in the treatment, no formula applicable to every case can be devised. The essentials of treatment today remain the same as during the time of Trudeau and other pioneers, and these essentials are rest of body and mind, good food and fresh air.

One of my professors, while lecturing to the class on tuberculosis said, "there are three essentials in the treatment of this disease—rest, rest, rest." And so today, rest continues to be the most important feature. But during the last twenty years, surgery has come to the rescue and brightened the path of the consumptive with another ray of sunlight. The ray of sunlight to which I refer, is the operation known as phrenicectomy.

Phrenicotomy was suggested by Stuertz in 1911, for the treatment of basal tuberculosis and bronchiectasis, and soon after used independently by Sauerbruch, who reported five cases in 1913. But the paralysis of the diaphragm which was the aim of this operation, was noted to fail in a percentage of the cases. This was later shown to be due to the presence of accessory phrenic nerves below the area of operation. Accessory phrenic nerves occur in twenty-five to thirty-five per cent of the cases according to Felix, who in 1922, proposed an exairesis of the phrenic nerve. Goetze objected to the phrenic exairesis of Felix, believing that there was danger of damaging important vascular structures or tearing into important structures in the mediastinum. Therefore, he proposed resecting a small portion of the nerve to the subclavius, as well as all accessory fibers between the phrenic and the cervical nerves.

In reviewing the literature of the operations done in this country, I find no

mention of damage to vascular structures due to an exairesis of the nerve, although as has been pointed out by Matson,[1] certain anomalies of the phrenic may occur whereby an exairesis may be fraught with danger. Some of his excellent illustrations show accessory branches to the phrenic encircling important vascular structures which could easily be damaged during an exairesis of the nerve. This makes it very important to avulse the nerve slowly and to cautiously observe for any evidencce of hemorrhage in the operative area.

Although this operation was at first recommended only for basal tuberculosis, experience has taught us that cavities in the apex can be closed and lesions in the upper part of the lung that are undergoing caseation often become quiescent following phrenicectomy.

Indications for the operation according to Alexander,[2] are:

1. In acute, highly febrile, progressive, predominately caseous types of tuberculosis, even when there is considerable activity in the better lung and when adhesions prevent the use of artificial pneumothorax and when extra pleural thoracoplasty is contraindicated.

2. For chronic types of disease, in which pneumothorax cannot be induced because of adhesions, and in which thoracoplasty is feared because the patient's general condition is too poor, or because the better lung is too much diseased.

3. For those moderately advanced or far advanced cases in which no definite contraindication to artificial pneumothorax or thoracoplasty exists, but in which phrenicectomy might be expected to effect good results without the prolonged treatment and complicating dangers of the one and the hazards of the other.

4. For those cases of early unilateral tuberculosis which do not show reasonable promise of becoming arrested by a continuation of sanatorium treatment.

It may often be used to advantage supplementary to artificial pneumothorax or thoracoplasty:

1. When partial or complete pneumothorax is not producing satisfactory results, phrenicotomy and pneumothorax may check the disease and make a thoracoplasty unnecessary, especially when there are basal adhesions and when adhesions of the lower lobe to the diaphragm are causing pain, irritative cough, vomiting or indigestion.

2. When it is feared that the lung will gradually come out in the presence of a pleural effusion and become adherent. From experiments on rabbits Balderry[3] found that in the presence of an artificially produced pleural effusion the diaphragm became fixed in the depressed position, but that when phrenicectomy was done before adhesions formed the diaphragm became fixed in the elevated position.

3. Another particularly valuable use for phrenicectomy is for persistent hemoptysis that pneumothorax does not succeed in checking because the part of the lung that is bleeding is held out by adhesions.

Alexander[2] and several others have recommended phrenicectomy supplementary to every artificial pneumothorax, claiming that the principal advantages to be gained are:

1. When the pneumothorax is discontinued the shrunken fibrotic lung will need to expand only enough to fill a thoracic cavity that has also been reduced in size and not one that is so much larger than the lung that the walled-off tubercles and closed cavities might be torn open. The lessened respiratory activity after phrenicectomy is further insurance against reactivation of the tuberculosis when the patient resumes active life.

2. Sauerbruch, Baer, Zadek and Ziegler report fewer and smaller effusions when pneumothorax and phrenicectomy are combined than when pneumothorax is used alone.

3. In case the patient prematurely abandons the pneumothorax, the high immobile diaphragm will forever serve as a partial substitute for the total compression of the abandoned pneumothorax and may be sufficient to maintain the gains already made.

Archibald[4] and others recommend phrenicectomy supplementary to extrapleural thoracoplasty. The special indications are:

1. To bring about sufficient improvement in a very sick patient so as to make later thoracoplasty a safe undertaking.

2. Preliminary to a thoracoplasty of only the upper ribs.

3. To increase the compression of the

lower lobe when it is markedly diseased and when there is special reason to stop diaphragmatic movements.

4. To increase the effect of the thoracoplasty in relieving the heart, trachea and other mediastinal structures that have been displaced far to the diseased side by the pull of shrinking intrapulmonary scar tissue.

Anatomy and physiology: the phrenic nerve arises chiefly from the fourth cervical nerve with a few filaments from the third and a branch from the fifth, although this branch is occasionally derived from the nerve to the subclavius. It descends to the root of the neck, running obliquely across the scalenus anticus. It then passes over the first part of the subclavian artery, between it and the subclavian vein, and, as it enters the thorax, crosses the internal mammary artery near its origin. Within the thorax it descends nearly vertically in front of the root of the lung and by the side of the pericardium, between it and the mediastinal portion of the pleura, to the diaphragm. Complete paralysis of half of a non-adherent diaphragm by interruption of both main and accessory phrenics causes that half to rise passively into the chest and to remain in the full expiratory position. The diaphragm remains practically immobile during respiration, thereby obtaining partial rest to the diseased lung.

THE OPERATION

The patient is given morphine gr. 1-6 and atropine gr. 1-150 by needle one-half hour before the operation. The skin and underlying tissues are infiltrated with a one-half per cent solution of novocain, 12 c.c. usually being a sufficient amount. A transverse incision about 6 centimeters in length is made about 6 centimeters above the clavicle beginning at the posterior border of the sternocleidomastoid muscle. The external jugular vein is usually encountered but it can be retracted or cut and ligated. After finding the nerve it is gently pinched with forceps and the patient practically always complains of pain in the shoulder and arm and occasionally in the region of the heart. The diaphragm can usually be felt and occasionally seen to contract. A few drops of novocain is then injected into the nerve, it is then separated from the surrounding structures, firmly grasped with an Ochsner or other suitable forcep, then cut and slowly extracted. It is very important to use slow and gentle traction, otherwise it frequently breaks and a sufficient portion of the nerve may not be destroyed to bring about the desired result.

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A BIOLOGIC CONCEPT OF DISEASE

Eli Moschcowitz, New York (Journal A. M. A., Aug. 27, 1932), states that, the more one sees of chronic disease, the more keenly one appreciates that what has been observed are not sharply defined genera but transitions of morbid states from one to another. One realizes that much of the differentation that has resulted from more refined observation and more detailed laboratory investigation has tended to confuse rather than to simply the issue and has caused the classification of disease to be more artificial than biologic. Too often diseases are classified according to a mere grouping of signs and symptoms, rather than in relation to a precise etiology or a well observed consistent pathogenesis. Many chronic diseases, like biologic species, present an evolution from the primitive or embryonic to a full-fledged form. It would be just as consistent to classify the earliest, the intermediate and the final phases of disease as separate entities as it would be to classify the tadpole and the frog as different species. Most of the confusion in classifying disease comes from an ignorance of the causes of disease. As long as the cause of many chronic maladies is unknown, so long will one be forced to classify disease on the basis of clinical differentials. This does not mean, however, that mere modifications of disease processes require separate classification. Time and again, conditions that are regarded as separate disease entities eventually prove to be only biologic sports or mutations. The author calls attention to the value of the biologic assay in evaluating disease and to the necessity for studying its evolution over more extensive periods than have been customary. Unfortunately, the number of diseases in which there are positive criteria or tests for diagnosis, such as the finding of the tubercle bacillus, are limited. For the others, until either a definite cause or a definite pathogenesis has been discovered, the only way to reach an understanding of the disease is by studying its evolution, both clinically and anatomically. Such a study, moreover, is necesary in order to determine the etiology of certain obscure conditions of a long life cycle. What, for instance, is the earliest phase of the leukemias? of pernicious anemia? of Gaucher's disease? of primary polycythemia? The determination of such origins may furnish an important clue to their causes.

A METHOD OF DETERMINING PERCENTAGE OF PARTIAL PERMANENT DISABILITY FOR THE OKLAHOMA STATE INDUSTRIAL COURT

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The physician and the industrial court: When an injured employee has sustained a disability of a permanent nature, the industrial courts expect the physician to determine the extent of disability. They depend on him to express in percentage the permanent or partial permanent loss, *i. e.*, he is to determine the functional ability less a disability.

This involves the physician in a matter that is without the usual sphere of his medical training. The average physician is not experienced in industrial court activities and the problem of interpreting loss of function in mathematical or monetary values is entirely foreign to his customary reasoning of prognostic determination. Since he probably has never done hard manual labor it is difficult for him to determine the ability of an injured laborer to perform his duties. The average physician is not accustomed to being definite. In his practice he deals constantly with uncertainties. He is well acquainted with, and often makes indefinite patronizing expressions, optimistic and sympathetic for the psychological benefit of the patient. When confronted, therefore, with a problem which has on the one side a claimant who expects monetary remuneration for damages to his body, and on the other side an employer who is forced by law to pay for such damages, the medical opinion immediately becomes valuable and all statements made must be such as to avoid prejudice and error.

It behooves the physician to be strictly impartial. However, testimony and opinions are rendered in every industrial court which show a wide variation in mathematical evaluations of percentages of partial disability. This cannot be remedied unless some definite system of reasoning can be evolved. It is the writer's contention that disability should be based on functional loss and not upon anatomical and physiological imperfections. The disability should be evaluated upon what the man cannot do because of anatomical and physiological inperfections, not upon the extent of their existence. For instance, pain, atrophy or shortening of an extremity, measure the disability only to the extent that they interfere with such functions as quickness of action, coordination of movements, strength or security . Thus, we would have to consider two elements in reasoning a determination of evaluation. First, the contributing factors of anatomical and physiological damage. Second, the functional loss resulting from these imperfections.

Contributing Factors: The contributing factors of a partial disability claim, *i. e.*, the anatomical and physiological deficiencies, for which an accident of industry is responsible are revealed by physical examination. For instance, a fractured bone that has healed in angulation necessarily disturbs mechanical perfection of the part. It may or may not interfere with use of the part in labor. If in the examiner's opinion it does interfere with function, the extent of this loss may be expressed in terms of percentage representing a limitation of the normal service of the part of the individual, and therefore, a diminished earning capacity.

Formulated Plan Necessary: In order to develop a method of determining the evaluation of permanent disabilities that is fair, it is necessary to formulate a plan whereby all the various factors comprising the disability may be analyzed and expressed as a percentage of loss. After considering the relative values of the individual loss of each factor, a summary then can be taken and the partial permanent disability of the entire part expressed in arithmetical value.

Functional Factors: Functional factors pertaining to the ability of an individual to use his limbs and body in ordinary manual labor at normal wage earning capacity are as follows:

1. Quickness of action.
2. Coordination of movements.
3. Strength.
4. Security.
5. Endurance.
6. Safety.
7. Ability to secure employment.

Quickness of Action — Alertness and rapidity of movement are essential. As the result of an injury the part may be lacking in this capacity and the loss of function would be termed "delayed action," *i. e.*, the resulting physiological or

anatomical handicap such as a deformity or stiffness will cause him to be slower than normal.

Coordination of Movements—is necessary for smoothness and steadiness of action. This may be termed dexterity or synchronizing of movements resulting in proficiency, deftness, and good control. The loss of this function would be termed as "awkwardness," or "clumsiness." For instance, because of a stiffness in the elbow the individual might reach quickly for an object and miss it or drop it.

Strength—physical energy and force of intensity is a very vital element of function. Loss of strength in a part causes the individual to make an unusual effort or strain to keep up with his work and although other functions are closely allied to this one function, it stands out as an important factor, the loss of which is noticeable, such as in the act of lifting, pinching or carrying, walking or standing. The loss of this function would be termed "weakness." This functional factor and that of endurance are often similar, but a distinction usually can be made.

Security—and confidence in use of the part is another very important function. Once a part of the body has been injured, soreness results and interruption of the habits of activity is slowly overcome. An organ of the body has not regained trustworthiness and reliability until it can be used without a conscious effort. Where this factor has been affected, it may be determined, "insecurity or uncertainty of movement, grasping, holding or stepping." For instance, in walking or running, an individual with a shortening of his leg would be constantly on guard to prevent making a misstep or falling.

Endurance—and vigor is a factor of work power that ofen depends upon the willingness of effort and persistency of the individual. He may seem to be able to work but can withstand the strain of work for a short period only. He, therefore, cannot continue and maintain his work uninterruptedly and cannot be kept on the job. Affection of this factor may be termed, "diminished endurance and vigor."

Safety—and ability to protect oneself and others while at work, is of great importance in hazardous occupations and may be greatly jeopardized by a disabled part of the body. Ability to jump quickly, danger of letting go a heavy object that would strike others, or failure to exercise the part in a normal manner while working with others, is a factor which would cause any superintendent to dismiss the individual. "Endangering safety," then, is a factor in the loss of function. It involves other factors but should be given its share of evaluation because it is not always a factor of loss, even when awkwardness or slowness is counted a loss.

Ability to Secure Employment—Physical fitness as a factor to securing employment is of major importance in considering the extent of disability. A deformity or a stiffness of a part decreases the competency and ability and will be detected by a company examining physician who is passing only 100 percent physically fit men. In times of distress, he will be the first to be laid off. Of course, many jobs are open which do not require physical examination, nevertheless this factor plays a big role in the ability of the individual to obtain work. "Interfering with securing employment," is, therefore, considered a loss of function. This function involves the other functional losses but is not always a factor of loss even where other functions are disturbed.

The functional factors of loss then are:

1. Delayed action, slowed up motions.
2. Awkwardness and clumsiness in grasping, pushing and holding.
3. Weakness of lifting power and muscular action.
4. Insecurity and instability in holding or pushing or pulling power.
5. Diminished endurance, vigor and vitality.
6. Lowered safety and protective ability.
7. Interference with chances for securing employment.

Rating of Functional Factors. It is reasonable that if each of these functional factors enter into the disability of the individual, then each should bear it's relation to the evaluated loss. No single factor alone is responsible for total disability. For instance, if the disabled member of the body is 100 per cent weak, quickness of action, coordination of movements, security and endurance are also affected, but not necessarily to the amount of 100 per cent in respect to performing ordinary manual labor. It

is necessary, therefore, that each of these factors should be given an evaluation according to its relative importance to the working capacity of the individual. The slowing up of activity and awkwardness are not important to the earning capacity as weakness in lifting power or the insecurity of holding, pulling or pushing power. The factors of diminished endurance and endangering safety are not to be rated quite as high as the damaging effect of lessening the individual's chance of securing future employment. The relative value of any of these factors will vary in many ways, in various cases. If each factor is given a percentage value and the sum of all factors is 100, then even though relative importance may vary, the percentage of loss of the part as a whole will be equitably estimated. There may be a difference of opinion on the individual functional values, but if each is a portion of 100%, the resulting permanent loss of each will total a proportional percentage.

The relative value of each of these functional factors of loss in respect to 100% incapacity may be generally rated as follows:

1. Delayed action	10%
2. Awkwardness	10%
3. Weakness	20%
4. Insecurity	20%
5. Diminished endurance	10%
6. Lowered safety factor	10%
7. Interfering with securing employment	20%
	100%

These functional factors may be rated differently by different individuals or they may vary with the part affected, as *e. g.*: in estimating the disability to the back, the functional factors of loss may be rated as follows:

1. Delayed action	10%
2. Diminished lifting power	20%
3. Awkwardness of incoordination	20%
4. Diminished endurance	20%
5. Interference with safety	10%
6. Interfering with securing employment	20%
	100%

Variations of Opinion: Some examiners might not agree on this evaluation of factors. They might feel, for instance, that the weakness should be valued at more than 20 per cent. If so, then some other function must be diminished in its relative value, because the total cannot be over 100 per cent. For instance, if weakness were considered 40 per cent in relation to the other functions, the value of each factor would be rated in their proportional value of the 100 per cent. A percentage of disability based on this form of reasoning, therefore, establishes a definite basis for its existence, so that if a physical examination reveals certain physiological and anatomical limitations, the effect on each functional factor can be estimated as the percentage of loss in respect to that particular capacity. The total percentages of loss will represent the partial loss of the part as a whole.

Illustrative case: Suppose it is found that an injury to the elbow has fully healed and on examination presents the following clinical picture:

The motion of flexion and extension is limited to an arc from full extension to 90 degrees flexion; other motions normal. The percentage of loss due to this physiological and anatomical limitation, as it contributes to each functional factor, would be estimated approximately as follows:

Delayed action: 25 per cent, because the alert and rapid movements of the arm would be cut down one-fourth that of normal, in respect to work.

Awkwardness: 25 per cent, because the coordination of movements and dexterity would be diminished at least this much.

Weakness: 15 per cent. This function would not necessarily be so great because an arm can be very strong, yet limited very greatly in motion.

Insecurity: 15 per cent, because there certainly would be a lack of confidence and interruption of previously formed reliability upon use of the arm.

Diminished Endurance: 15 per cent, because the necessity of increased effort in use of the arm would produce early tiring on the part of the individual.

Endangering safety while at work: 10 per cent, because in a hazardous occupation he would be more apt to let go an object or fail to grasp an object in his protection.

Interfering with securing employment: 15 per cent, because an employer or an examining physician for an employer would consider the handicap as a depreciation of the desirability or worth of the individual's working ability.

Evaluation of the partial loss of the part affected would be determined then by summing up these contributions to a

partial loss in respect to each functional factor.

25% of 10% (delayed action evaluation)	2.5
25% of 10% (awkwardness evaluation)	2.5
15% of 20% (weakness evaluation)	3.0
15% of 20% (insecurity evaluation)	3.0
15% of 10% (diminished endurance evaluation)	1.5
15% of 20% (interfering with securing employment evaluation)	3.0
	15.5

The partial loss to the entire arm, therefore, would be 15.5%.

Additional disabilities: If the clinical picture presented additional physiological or anatomical limitations, the effect of these would be estimated in percentage of loss to the remaining value of the part after deducting the first element of disability. For example, suppose that in addition to the partial ankylosis of the elbow, the examination reveals a fracture of the humerus which has healed in angulation of 20 degrees, with good union. The percentage of loss to the arm, as it contributes to each functional factor, would be approximately as follows:

Delayed action	0%
Awkwardness	5%
Weakness	10%
Insecurity	5%
Diminished endurance	5%
Lowered safety factor	0%
Interfering with securing employment	5%

With these estimates made, mathematical calculation is as follows:

0% of 10% (delayed action)	0
5% of 10% (awkwardness)	.5
10% of 20% (weakness)	2.0
5% of 20% (insecurity)	1.0
5% of 10% (diminished endurance)	.5
0% of 10% (lowered safety)	0
5% of 20% (interfering with securing employment	1.0
Partial loss to the arm	5.0

Calculation of additional disability: Each loss factor must be deduced according to percentage rules.

The total of the partial loss to the arm, as a result of the fractured humerus, is 5%. However, since the loss to the arm from the elbow injury is 15.5% there is only a balance of an 84.5% arm on which to estimate an additional 5 per cent loss. 5 per cent of 84.5 is 4.22.

15.5% plus 4.22% gives a total of 19.72% disability to the arm as a result of the two factors of loss.

Specific losses: The compensation laws specify a certain number of weeks or a percentage of loss for certain individual members or portion of members of the body. A specified amount, for instance, is provided for total loss of use of the entire body, the arm, leg, hand, thumb and each finger and toe.

Where the law stipulates awards in numbers of weeks, the values may be reduced to percentage, to facilitate mathematical calculation. The value of the arm as established by the Oklahoma law is 250 weeks or 50 per cent of the entire body, which has a value of 500 weeks for total permanent disability. The value of the hand is 200 weeks or 80 per cent of the entire arm. The value of the fingers is: thumb 60 weeks; first finger 35 weeks; second finger 30 weeks; third finger 20 weeks and fourth finger 15 weeks. The combined value of the fingers and thumb is 160 weeks or 64 per cent of the whole arm and 80 per cent of the whole hand. It is sometimes necessary to know the value of the remainder of the hand and forearm, exclusive of the fingers. This is obtained by deducting the combined value of the thumb and fingers, which is 160 weeks, from the value of the whole hand, which is 200 weeks. 200 minus 160 is 40. Therefore the value of the hand and forearm in relation to the hand only would be 40 weeks or 20 per cent of the hand. The value of this part of the hand to the whole arm would be 20 per cent of the value of the whole hand to the value of the whole arm, which is 80 per cent. 20% of 80 is 16%. A disability to the hand of a permanent nature affects, from the functional standpoint, the entire arm, yet, if it is below the elbow, the legal point of view permits consideration of loss to the hand hand and arm, yet from the legal standpoint, only the specified value of the digit it allowed.

A disability to a finger, therefore, must be estimated as a percentage of loss to the finger only. If loss of the finger is to be expressed in per cent of the loss to the entire arm, the estimate is based on the value of the finger to the arm. (See Table No. I). For example, if there is a 20 per cent loss to the index finer, then the loss to the arm is only 2.8 per cent, because the index finger is 14 per cent of the arm.

In a similar manner, a loss of 25 per cent of the hand will be but 20 per cent loss to the arm as a whole. (See Table No. II), because the hand is but 80 per cent of 250 weeks, the value of the entire arm.

Where the forearm or palm is involved but the fingers and thumb action is in no way disabled, then the value of the forearm and hand to the whole hand must be considered with the finger and thumb value excluded. If a 25 per cent disability is limited strictly to use of the forearm, it

TABLE NO. I.

COMPARATIVE VALUE OF PARTS OF THE ARM

Loss of a thumb	60 wks;	24% loss of arm 250 wks;	30 % loss of hand 200 wks.
Loss of 1st or index finger	35 wks;	14% loss of arm 250 wks;	17.5% loss of hand 200 wks.
Loss of 2nd finger	25 wks;	10% loss of arm 250 wks;	12.5% loss of hand 200 wks.
Loss of 3rd or ring finger	20 wks;	8% loss of arm 250 wks;	10 % loss of hand 200 wks.
Loss of 4th or little finger	15 wks;	6% loss of arm 250 wks;	7.5% loss of hand 200 wks.
Loss of a hand	200 wks;	80% loss of arm 250 wks;	100 % loss of hand 200 wks.
Loss of all fingers exclusive of forearm	160 wks;	64% loss of arm 250 wks;	80 % loss of hand 200 wks.
Loss of forearm exclusive of fingers	40 wks;	16% loss of arm 250 wks;	20 % loss of hand 200 wks.
Loss of an arm	250 wks;	100% loss of arm 250 wks;	50 % loss of body 500 wks.

TABLE NO. II.

COMPARATIVE VALUE OF PARTS OF THE LEG

Loss of first toe	30 wks;	17.2% loss of leg 175 wks;	20 % loss of foot 150 wks.
Loss of any other toe	10 wks;	5.4% loss of leg 175 wks;	6.6% loss of foot 150 wks.
Loss of foot	150 wks;	80 % loss of leg 175 wks;	100 % loss of foot 150 wks.
Loss of toes, exclusive of foot	70 wks;	40 % loss of leg 175 wks;	60 % loss of foot 150 wks.
Loss of foot, exclusive of toes	80 wks;	45.7% loss of leg 175 wks;	53.3% loss of foot 150 wks.
Loss of leg	175 wks;	100 % loss of leg 175 wks;	35 % loss of body 500 wks.

would be estimated as 25 per cent of 20 (See Table No. I), or a value of only 5 per cent loss to the hand as a whole. To the entire arm it would be 25 per cent of 16 or 4 per cent loss.

MULTIPLE DISABILITIES RESTRICTED BY SPECIFIC LEGAL EVALUATION

Losses as usually specified by compensation laws are based on anatomical and physiological deficiencies and not on functional capacity. Where there are multiple lesions, therefore, the evaluation often will be found to be inadequate or too generous. If the functional standard is adhered to as a basis of estimate, a more just evaluation may be calculated. For instance, a lesion to the upper arm, another to the forearm and another to a finger, each separately may be evaluated to a percentage of loss, the sum of which would exceed 100 per cent of the arm.

For example, suppose it is estimated that because of a fracture of the humerus, involving the ulnar nerve, there is a loss to the entire arm of 60 per cent. A complete ankylosis of the elbow might be estimated at 50 per cent loss of the entire arm, and a partially ankylosed thumb might be evaluated as 10 per cent of the entire arm. The sum of these evaluations would be 120 per cent. However, in reality, there still may be some function left in the arm.

The total loss from multiple lesions should not be greater than the functional good of the part less the functional loss. By giving each part affected its percentage of value to the arm as a whole and estimating the percentage of each part on this basis, the relative functional loss will be in proportion as it affects the arm as a whole. For example, a series of lesions to the arm may be as follows:

1. An injury to the index finger causing a loss of 25 per cent.

2. An injury to the forearm causing a loss to the hand of 50 per cent.

3. An injury to the upper arm causing a loss to the arm of 40 per cent.

What is the loss to the entire arm? It is obvious that it cannot be estimated by adding the specific losses, as this would total 115 per cent.

Since the index finger is valued at 14 per cent of the entire arm, (See Table No. I), 25% of 14 is 3.5%. So that this lesion will contribute 3.5 per cent to the entire arm loss. This loss must be deducted from 100 per cent to obtain the amount of value of the remainder of the arm upon which to make further deductions. 100% less 3.5% leaves a balance of 96.5% good arm. Since the hand is valued at 80 per cent of the arm, then 50 per cent loss of the hand would be 40 per cent loss to the entire arm. 40 per cent loss of the affected arm then would be 40% of 96.5 or 38.6% loss. Subtracting 38.6% from 96.5% leaves a balance of 57.9% arm. An additional 40 per cent loss due to the upper arm injury would have to come from the remaining 57.9 per cent arm. 40% of 57.9 is 23.16%. 57.9% minus 23.16% leaves a balance of 34.74% arm. Subtracting 34.74% from 100% then, leaves a loss from the multiply influences the function of the entire disabilities of 65.26%. The individual losses then are as follows:

Loss of the arm due to finger injury	3.5 %
Loss of the arm due to forearm injury	38.6 %
Loss of arm due to upper arm injury	23.16%
Total loss to the arm	65.26%

Illustrative case: Mr. R. A., reopened his case before the Industrial Commission, claiming a change for the worse. Two years prior to the present claim, he had

received a settlement of 50 per cent loss of use of his foot. On examination it was found that he had developed atrophy of the thigh and stiffness of the knee for which an estimate was made of 10 per cent partial permanent loss of the leg. On cross examination the claimant's attorney insisted that this added disability should give the claimant a 60 per cent loss of the entire leg. This could not be possible from the mathematical standpoint and it was estimated as follows:

50 per cent loss of the foot is only 40 per cent loss to the leg as a whole, and he had been paid this amount, so that additional disability should not include what has already been counted as lost and paid for. He, therefore, only had a 60 per cent leg upon which to estimate further disability. 10 per cent of 60 is 6. Therefore, the additional loss is 6 per cent, instead of 10. His partial permanent disability then would be 40 per cent, his former loss, plus 6 per cent, his present loss, or a total of 46 per cent loss.

CONCLUSIONS

The writer has been stimulated to study out some definite ideas on this subject because it seems that dealings with industrial claims have come to be a part of the physician's duties and that the profession should take some steps to place it on just as honorable and scientific basis as any other part of medical practice.

It is hoped that the suggestions here developed will inspire others to continue the study.

DISCUSSION

Question: I should like to ask if Dr. McBride has tried this out on the Industrial Commission, and with what results.

Dr. McBride: I had about a three-hour session with the commission one day and the members suggested that as a profession we ought to have some common understanding of this thing and some method whereby reasoning could be used in testimony instead of arbitrary statements. They have used my method of estimating disabilities in a number of instances, to my knowledge. The compensation law is based on the old European law that has just been handed down. There is not much of a fundamental basis for their awards and they have just been copied from state to state in this country. The law is based on physical and anatomical loss, but the medical profession must determine the percentage of loss. In testimony, some physicians have one impression relative to his experience and others have their impressions. I think it is our business to testify on the amount of function. We do not know so much about wages and employment. The layman knows nothing about function, so that if we can analyze function and reason it out on that basis we will at least remain in our professional domain.

INTRAVENOUS TREATMENT IN EXPERIMENTAL TRICHINIASIS

J. J. Miller, Jr., San Francisco; O. R. McCoy and W. L. Bradford, Rochester, N. Y. (Journal A. M. A., April 9, 1932), made an attempt to test the efficacy of several therapeutic agents in the parenteral treatment of trichiniasis. Experimentally infected rabbits were given intravenous injections of neoarsphenamine, antimony and potassium tartrate, acriflavine base, rivanol, gentian violet, metaphen, and compound solution of iodine (Lugol's solution). Experimentally infected rats were treated with intraperitoneal injections of sodium iodide. In no instance was any demonstrable therapeutic effect obtained. On the basis of these experiments, the authors question the therapeutic rationable of the occasional injection of such drugs as the arsphenamine derivatives and antimony and potassium tartrate in the treatment of human trichiniasis.

SURGICAL TREATMENT OF PROGRESSIVE EXOPHTHALMOS FOLLOWING THYROIDECTOMY

According to Howard C. Naffziger and O. W. Jones, Jr., San Francisco (Journal A. M. A., Aug. 20, 1932), progressive exophthalmos following thyroidectomy is of mechanical origin and is the result of a pronounced swelling of the extra-ocular muscles. Grossly the muscles are from three to eight times their normal size. Microscopic study shows edema, round cell infiltration—often in a perivascular pattern—loss of muscle architecture, increase of fibrous tissue with areas of hyalinization, and fragmentation and destruction of muscle fibers. The authors outline an operative treatment that is not intended as a cosmetic operation, nor is it advised for those persons who have simply a persistent, stationary exophthalmos following thyroidectomy. The precedure is for those who have a progressive exophthalmos, usually with limitation of movements of the globe of the eye, with or without changes in the optic nerves and in vision. It is pointed out that, in certain instances, vision fails even though the cornea is clear, and the opthalmoscopic examination reveals a normal fundus. The principle of the operation is that of decompression of the orbit and the optic formamen in order to give adequate space for the increased orbital content and the constricted optic nerve. One well may ask at this stage, "Is such a procedure sufficient to halt the pathologic process that is occurring?" The author can only answer that, up to date, it has seemed to be entirely adequate; in his patients there has been no recurrence, in all of them vision has been restored and there has been recession of the exophthalmos.

THE JOURNAL
OF THE
Oklahoma State Medical Association

Issued Monthly at Muskogee, Oklahoma, under direction of the Council.

VOL. XXVI MARCH, 1933. No. 3

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Entered at the Post Office at Muskogee, Oklahoma, as second-class mail matter July 28, 1912.

This is the official Journal of the Oklahoma State Medical Association. All communications should be addressed to The Journal of the Oklahoma State Medical Association, 525-526 Commercial National Bank Building, Muskogee, Oklahoma. $4.00 per year; 40c per copy.

The editorial department is not responsible for the opinions expressed in the original articles of contributors.

Reprints of original articles will be supplied at actual cost provided requests for them is attached to manuscripts or made in sufficient time before publication.

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Failure to receive The Journal should call for immediate notification of the editor, 810 Manhattan, Building, Muskogee, Oklahoma.

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PRINTED BY HOFFMAN-SPEED PRINTING CO., MUSKOGEE

EDITORIAL

COMMITTEEMEN FOR THE ANNUAL SESSION, OKLAHOMA CITY, MAY 15, 16, 17, 1933

Dr. Henry H. Turner, General Chairman of the above meeting, has reported the following selections as committeemen to handle the various phases of the meeting:

Advisory Committee:

Dr. Arthur W. White, Chairman
Dr. L. J. Moorman
Dr. LeRoy Long
Dr. A. L. Blesh
Dr. J. M. Alford
Dr. E. S. Ferguson
Dr. A. B. Chase.

The Chairmen of the other committees are as follows:

Finance	Dr. Bert F. Keltz
Entertainment	Dr. Rex Bolend
Hotels	Dr. R. O. Early
Registration	Dr. J. C. MacDonald
Golf	Dr. Wendell Long
Scientific Exhibits	Dr. Floyd Keller

OUR ANNUAL SESSION, SOME OF ITS OBJECTS

It is believed that attention should be called to some of the objects and practices carried out by our annual session. Among these are:

(a) It is hoped no member will undertake to appear upon the program twice. If that does occur one of the appearances will have to be cut off. This occasionally happens and is obviously unfair to many other members who may also wish to read a paper before some of the sections.

(b) Please remember that matters of business are considered by the Council only; the House of Delegates has its hands full considering reports and other matters. In any event physicians desiring to place matters before either of these bodies should have them well prepared in advance, with no uncertainty attached to their meaning. This saves time, which is a very valuable thing.

(c) As before, both the Council and House of Delegates will meet on the first day of the meeting, May 15, 1933.

(d) As we have persistently requested heretofore, it is not only a courtesy but a duty of those who wish to read papers, to send a copy of their paper, or at least a thorough abstract of the contents of the paper to the person who may open the discussion of it.

(e) This year every feature of the meeting will be held under one roof and, so far as we know, upon two floors. The scientific and commercial exhibits and the registration and probably the Surgical Section will meet on the top floor of the Hotel Skirvin. It goes without saying that each member attending should register. Oklahoma holds, or has held, heretofore, a very high rate of attendance, considering the number of its members, but each year a certain number fail to register —that gives us somewhat of a black eye, so this year it is hoped that every attendant will register.

(f) Do not come to the meeting without first having your membership card and knowing that you are in good standing. Only those upon the printed roster, prepared in advance, are eligible for registration. This saves embarrassment on both sides, for the physician as well as the registrars. It also saves a great deal of otherwise wasted time.

SURGICAL CASES—MAKE YOUR ASSERTION POSITIVE

It is well known that in the minds of many people there is an unnecessary dread of surgical operations, the dread as a rule being all out of proportion to the work to be performed. Often these cases have been misadvised or poorly advised by the attending physician, and, as a rule, the attending physican, not the surgeon, first advises and treats the case for a more or less indefinite time. It should be remembered that practically all operations, if the surgeon is the master of the technic he should be, amount to little or nothing from the standpoint of pain or danger. Under no circumstances should a physician leave a case to a "surgeon" merely because they happen to be good friends—certainly the case should go to the best hands. Every general practitioner should bear in mind the possibilities of abdominal conditions. Most of these, if taken and handled early, amount to nothing, while procrastination, poultices, ice bags, and symptomatic medication often bring the case to a fatality or to the brink of fatality. Therefore, the attending physician should be able to fairly early make up his mind that "this is a surgical case," which is not a dangerous situation, but has grave possibilities, and should be referred to a competent surgeon at the earliest moment. It goes without saying that these cases belong in a hospital, and every hour of delay further endangers the patient. It is no reflection for a physician to call in a consultant. This consultant may find the case, instead of being appendicitis, gall bladder disease, duodenal, etc., to be one of an overlooked pneumonia. These are by no means uncommon.

The moral to all this is "be up on your toes."

—o—

STOPPING THE HURRY-UP CANDIDATE FOR MEDICAL LICENSURE

American students entering European medical colleges after the academic year 1932-1933, shall not be admitted to examination by any state licensing board, who do not, before beginning the course in such medical college, secure from a state board of medical examiners, or other competent state authority, a certificate endorsed by the Association of American American Medical Colleges or the Council on Medical Education and Hospitals of the A. M. A., showing that he has met the premedical educational requirements prescribed by the before mentioned associations, if the Federation of State Medical Boards of the United States, has any power in the matter. This is the gist of a decision of the Federation which met in Chicago December 18th. It is based upon the information and belief of the Federation that there are now, in Europe 1,500 "low-grade" American students, soon to be graduated "without clinical advantages, interneship, examination for license and other privileges given by such colleges to their native graduates—and returned to the United States for licensure and practice. It is the purpose of the Federation to stop the migration to European medical colleges of American students of sub-standard, premedical attainment, who are taking advantage of an opportunity to get the bare degree and return to this country to practice."

This is correct. It is obviously unfair, and discrimnatory, to the student of American medical colleges, required to put in four long years of his time, followed by an interneship of at least one year, to find himself confronted with, and in competition with, possibly a former fellow student, who simply packed up his grip, evaded certain reasonable premedical requirements of this country, and returns with a degree from some European medical school, technically covered by all the rights and privileges surrounding a student who has passed through years of the necessary and practical requirements demanded of the American student.

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Editorial Notes—Personal and General

DR. J. A. JONES, Tonkawa, is reported ill.

THE HYGEIA COMMITTEE of the Oklahoma County Woman's Auxiliary gave the third of a series of "Silver Teas" on February 15th, the proceeds to go for more Hygeia subscriptions.

THE DALLAS SOUTHERN CLINICAL SOCIETY will meet in Dallas, headquarters, Baker Hotel, March 27, 31, 1933. A number of distinguished guests from generally over the United States will be present.

CLEVELAND COUNTY MEDICAL SOCIETY elected the following officers for 1933: President, Dr. Carl T. Steen, Norman; Vice President, Dr. Eleanora Schmidt, Norman; Secretary-Treasurer, Dr. D. G. Willard, Norman.

KAY COUNTY MEDICAL SOCIETY met in January and elected the following officers for

1933: President, Dr. Merle C. Clift, Blackwell; Vice President, Dr. W. S. White, Blackwell; Secretary-Treasurer, Dr. L. H. Becker, Blackwell.

THE OKLAHOMA COUNTY WOMAN'S AUXILIARY to the Oklahoma State Medical Association, held their January meeting at the Oklahoma Club. Sewing for the Crippled Children's Hospital was the main feature of the meeting.

GRADY COUNTY MEDICAL SOCIETY elected the following officers at their January meeting to serve for 1933: President, Dr. J. F. Renegar, Tuttle; First Vice President, Dr. C. P. Mitchell, Chickasha; Second Vice President, Dr. C. P. Cox, Ninnekah; Secretary-Treasurer, Dr. L. E. Woods, Chickasha; Delegate, Dr. Roy E. Emanuel, Chickasha; Censor, Dr. D. S. Downey, Chickasha.

OKMULGEE COUNTY MEDICAL SOCIETY elected the following officers for 1933: President, Dr. I. W. Bollinger, Henryetta; Vice President, Dr. G. A. Kilpatrick, Henryetta; Secretary-Treasurer, Dr. M. B. Glismann, Okmulgee; Censor, Dr. W. M. Cott, Okmulgee; Delegates, Drs. W. W. Stark, Okmulgee, and G. Y. McKinney, Henryetta; Alternates, Drs. J. J. C. Rembert, Okmulgee, and J. P. Nelson, Schulter.

The January meeting was held at Henryetta, January 23rd. Dr. J. E. Hughes, Shawnee, lectured and showed his movie film of "A Hunting Trip to Alaska."

MUSKOGEE COUNTY MEDICAL SOCIETY held its regular meeting February 13th. Twenty-five members were present. Dr. C. V. Rice, reported a case of Atypical Meningitis. The following program was rendered:

1. "Postmortem Ceserean Section,"—Dr. C. S. Neer, Vinita.

2. "Pneumothorax in Tuberculosis,'"—Dr. J. T. Woodburn, Muskogee.

3. "Venous Pressure and Its Clinical Significance—Dr. H. R. Rothman, U. S. Veterans' Hospital, Muskogee.

4. "Pertinent Points on Muskogee County Medical Society in its Relationship to Organized Medicine and Its Membership,"—Dr. F. G. Dorwart, Muskogee.

"COUNTERFEIT" COURSES FOR GRADE 'A' SCHOOLS

The American Medical Association calls attention to the fact that there is being widely distributed an announcement of the Illinois College of Physicians and Surgeons, 20 North Ashland Boulevard, Chicago, which institution is offering courses pretending to fit one for requirements of class 'A' medical schools. The only organization, so far as physicians and surgeons are concerned, which issues these requirements is the Council on Medical Education and Hospitals of the American Medical Association. It seems the aggregation, above noted, is conducted by a group of chiropractors and does not even remotely approach the standards of a class A medical school.

This warning is published for the information of possible gullible students.

DOCTOR E. D. MABRY

Dr. E. D. Mabry, 58 year old, Oklahoma City physician, died January 25th, 1933, at the Polyclinic Hospital. His death followed some time after an operation which was necessitated by results of X-ray burns, first suffered when he experimented and used one of the first X-ray machines in the state of Texas, many years ago. Dr. Mabry was born in Burnett, Texas, February 27, 1874, the son of J. J. Mabry and Fannie Hoover Mabry. He graduated from Burnett, Texas, High School at the age of 16 and the next year began his studies in Washington University, School of Medicine, St. Louis, Missouri. He graduated there with honors as one of its youngest graduates in the class of 1895. After that he practiced his profession in Llano and Beaumont, Texas, and came to Oklahoma a few years ago, located in Oklahoma City, about two years ago in order to receive treatments for X-ray burns. He has taken post graduate work from time to time both in New York and in Chicago. He was associated with and active in the Medical Association of the localities in which he practiced and at the time of his death was a member of the American and the Oklahoma State and County Medical Associations.

Dr. Mabry was known as a physician of the "old school," insofar as his idealism, devotion to his patients, and loyalty to his profession were concerned. However he continued to keep abreast of the recent advances in medicine. Dr. Mabry is survived by his wife and two daughters, Mrs. O. A. Brewer, of Hugo, and Mrs Basil Wagoner of Chandler.

Dr. Mabry was laid to rest in the Fairlawn Mausoleum, Oklahoma City, Oklahoma.

DOSTOR JOHN Z. MRAZ

"In Memorium"

Dr. John Z. Mraz, age 50, died in Oklahoma City, Oklahoma, on January 12th, 1933, of tuberculosis of lower ileum and cecum, after an illness of several months. He was born in Riverside, Iowa, but moved to the city of Chicago with his parents when he was but a few years of age. There he received his preliminary education and graduated from the Rush Medical College in class of 1903. Soon after his graduation in medicine he located at Prague, Oklahoma, where he engaged in general practice until 1914, when he moved to Oklahoma City. There he became associated with Dr. A. L. Blesh in surgery, devoting his time to the field of genito urinary surgery.

When this country entered the World war Doctor Mraz joined the Oklahoma National Guard and in June, 1917, was sent to Ft. Leavenworth for training. He served overseas in capacity of Commander of Field

Hospital, saw action in both Meuse Argonne and St. Mihiel offensives and was discharged in July, 1919. He immediately returned to his former associates in practice in Oklahoma City. Continuing his associations with the group that had been formed with Doctor Blesh as the nucleus. In the fall of 1919, this group associated themselves together in partnership as the Oklahoma City Clinic, which purchased the Wesley Hospital from Doctor Camp in September, 1919. He remained as member of the clinic and on the active staff of Wesley Hospital until the time of his death. Besides his special work in charge of genito urinary surgery, he gradually perfected himself in general surgery through his association with Doctor Blesh. so that for some years he carried on with his special work a great deal of the general surgery of the clinic. As to his ability in surgery and especially in the field of genito urinary surgery, one can only say that he had the confidence of one who knows he is master of his subject. Extremely careful and methodical in his work, with his long and fairly extensive experience, no situation arose but what he handled with the skill of an expert.

His personality stood him in good stead on many occasions. He could face any situation with an equanimity which was the envy of his associates. An extensive and profound reader, he had gradually evolved a philosophy of life that was extremely tolerant of the many short comings of human nature. In religion his views were liberal.

The demands on his time for his work in his practice and the many committees and meetings to which he gave a considerable portion of his time, kept him extremely busy all the time. His home, his wife and children were ever uppermost in his mind however, and nothing gave him greater pleasure than motoring with his family on holidays or week ends.

He is survived by his widow, Betsy—nee Besty May, formerly of Wilkes Barre, Pa., whom he married in 1918, and two children, John, age 9 and Milada age 6.

DOCTOR CHARLES D. FREDERICK O'HERN

Dr. C. D. F. O'Hern, 54 year old physician of Tulsa, died in December from an attack of heart disease.

He was born in Hinsdale, New York, in 1878, but moved with his family when still a boy, to DuBoise, Pennsylvania. He attended the University of Maryland at Baltimore and did postgraduate work in Vienna.

He is survived by his widow and three sons, and two brothers.

Burial was in the Catholic cemetery.

DOCTOR GAYFREE ELLISON

Dr. Gayfree Ellison, Norman, died December 22, 1932.

He was born at Sheridan, Kansas, October 28, 1875. His preliminary education was obtained at Lindsborg, Kansas. He graduated from Rush Medical College, Chicago, April, 1903. Prior to coming to Oklahoma City, where he practiced from 1906 to 1910, Dr. Ellison was associated with Dr. O. A. Ochsner at the Augustana Hospital, Chicago.

Dr. Ellison has been director of the health service department and professor of epidemic subjects, at Oklahoma University for the past 22 years.

He is survived by his wife and two children.

PHYSOSTIGMINE, A PERISTALTIC STIMULANT

According to P. F. Butler and Max Ritvo, Boston (Journal A. M. A., Oct. 15, 1932), physostigmine increases gastric tonus and peristalsis. This drug is of great benefit in the roentgen examination of the stomach in patients with absent or sluggish peristalsis, marked atonicity or spasm. Physostigmine enables the roentgenologist to complete his studies of the stomach in a much shorter time and with a greater degree of accuracy in the aforementioned types of cases. The average dosage is 1-25 grain (2.6 mg.) orally, the drug being effective when given by mouth. This obviates the need of injection methods. The administration of physostigmine does not, as a rule, interfere with the routine roentgen studies of the gastro-intestinal tract. Reactions are but rarely encountered, and since atropine is a physiologic antidote to physostigmine, toxic manifestations are controlled without difficulty.

OLFACTORY DISTURBANCES

Ernest M. Seydell, Wichita, Kan. (Journal A. M. A., Aug. 20, 1932), calls attention to the fact that in patients with olfactory disturbances it is advisable to make tests of the acuity of the sense of smell, nasal taste, nasal tactile sense and the gustatory sense. Careful olfactometric examinations followed, when possible, by accurate postmortem examinations will extend the knowledge of olfaction. The cooperation of surgeons who come in contact with numerous skull fractures should be sought to assist in this problem. The intravenous injection of smell substances appears to be valuable in the differentiation between the respiratory and the essential anosmias, and in the treatment of the peripheral parosmias. Since the cocosmias and the peripheral parosmias are of a benign nature, it is essential that great care be taken in differentiating them from the central parosmias and hallucinations which point toward some serious cerebral disturbances.

ABSTRACTS ◆ REVIEWS ◆ COMMENTS AND CORRESPONDENCE

SURGERY AND GYNECOLOGY

Abstracts, Reviews and Comments from LeRoy Long Clinic

714 Medical Arts Bldg., Oklahoma City

A Variety of Firm Post-Traumatic Oedema and Oedema of Therapeutic Origin (Une Variete d'Oedeme Dur Post-Traumatique: L'Oedeme d'Origine Therapeutique), G. Metinet, LaPress Medicale, Feb. 1, 1933.

Secretan, of Lausanne, first described firm post-traumatic oedema. It is generally on the dorsum of the hand, and follows a relatively mild trauma. It is sometimes accompanied by decalcification of the subjacent skeletal structures. It is often obstinate.

Three types may be observed:

1. Simulation. Cases have been reported by Busacchi where the oedema was due to self inflicted repeated mild trauma of the back of the hand. Certain soldiers admitted that the oedema was produced by repeatedly striking the areas over the joints with the opposite fist.

2. Vaso-Motor Troubles of Sympathetic Origin. Reference is made to the work of Leriche who reported that firm oedema due to this cause rapidly disappears after periarterial sympathectomy.

3. Attenuated Infections of the Lymphatic System. Cases of this character have been reported by Imbert who regards them as "a sort of infectious elephantiasis" (une sorte d' elephantiasis infectieux).

A very interesting part of the report is the author's conclusion that there is a fourth type of firm oedema which is produced and kept up by frequent local hot baths after a trauma, especially a trauma of the extremity of a member (et particulierement d' un traumatisme portant sur l'extremite d' un membre)—hand, for example. The condition is usually noticed where the hands have been frequently held for sometime and repeatedly in water as hot has could be borne by the patient. The region becomes infiltrated, oedematous, thickened, indurated, with stiffness of the points and limited play of the tendons. **And the hot water is the sole cause** (Et la seule coupable de ce facheux etat est l' eau trop chaude). The treatment is to simply replace the hot baths by tepid baths when the oedema will disappear, and articulations and tendons will become normal.

—LeRoy Long.

The Hazards of Intraperitoneal Injections by Samuel F. Ravenel, M.D., A. M. A. Journal, February 18, 1933.

Intraperitoneal injections of normal saline have been employed extensively since 1918, in the treatment of dehydration in infants. More recently glucose, diphtheria antitoxin, neoarsphenamine, iron and blood have been given by this route.

This route permits more rapid introduction into the body of relatively larger quantities of fluid than is possible by hypodermoclysis. It 's also easier than venipuncture. This explains its popularity.

It has been said to be difficult to perforate the intestine of living animals or of cadavers. There is a widespread conviction that intraperitoneal injection is entirely safe in the absence of marked abdominal distention or of inflammatory disease of the abdomen. It is the practice in some large clinics and on the part of many pediatricians to ignore moderate tympanites when fluids are needed.

That there is danger in this route of administration is borne out by cases reported by this author.

In one case, perforation of the intestine occurred during intraperitoneal injection of saline solution. In this case there were no adhesions between intestine and the parietal peritoneum. The usual precautions of using a dull needle and of pinching up of the tissues were observed. During the last week of life moderate abdominal distention existed. One must infer from this case that tympanites constitutes an absolute contraindication of intraperitoneal injections.

In two instances, incompatible blood administered intraperitoneally produced violent local and general reactions (some authors have considered typing or cross agglutination unnecessary preliminary to this method of blood transfusion).

In two cases, serious hemorrhage resulted from the "obliterated" hypogastric artery during the course of intraperitoneal injections in very young infants.

The author's conclusions with which I agree are as follows:

1. Abdominal distention is an absolute contraindication to intraperitoneal injection.

2. The introduction of unmatched or incompatible blood into the peritoneal cavity is dangerous and unwarranted. Cross agglutination before each such transfusion is necessary, irrespective of whether or not the same donor previously has been used with success.

3. In premature or very young infants, intraperitoneal injection in the midline below the umbilicus may produce serious hemorrhage by wounding the "obliterated" hypogastric artery.

4. Intraperitoneal injection, while affording the most satisfactory method of administering fluids parenterally to infants, is not so entirely devoid of danger as is generally believed.

—LeRoy D. Long.

Prevention of Chills Following Transfusion of Citrated Blood. Richard Lewisohn, M.D., and Nathan Rosenthal, M.D., New York. A. M. A. Journal, Volume 100, No. 7, Feb. 18, 1933.

This simple and easy procedure was suggested by Lewisohn eigtheen years ago and since then blood transfusion has been put within the reach of every physician and surgeon. When this method was introduced, many objections were voiced against its clinical efficacy. It was claimed that the vitality of the red cells and white cells was impaired by the mixture of blood with sodium citrate. Furthermore, many clinicians felt that the introduction of an anti-coagulant was contraindicated in hemorrhagic diseases and other forms of blood dyscrasia. It has been fairly well proven that these objections are purely theoretical and that citrated blood has the same clinical value as unmodified blood, not only in cases of shock and hemorrhage, but in every form of blood.

Unquestionably a chill following a transfusion may be a serious complication. In many instances transfusions are given to patients who are very ill. In such a case, one should employ the method that produces the least number of chills. On this basis a careful clinician may prefer non-citrated blood.

However, these authors have compared large series of cases in which non-citrated blood was used, with a similar series of transfusions by the citrate method and have shown that there is no difference in the number of chills if equal care as to details of technique is applied.

The fact that chills do not always follow the use of citrated blood is indicative of the fact that the minute quantity of sodium citrate is not the cause of these chills. Also, it is a fact that in the earliest transfusions there were fewer chills than at present, and this is accounted for by the author who says that he personally attended to the preparation of the vessels, tubing and solution. In recent years with the enormous number of transfusions done by many different individuals in the hospitals there has not been standardization of technique in preparing the apparatus to be used. The preparation of the instruments (cannulas, glassware and the like) used for intravenous therapy was too lax and required standardization.

The solution of this important problem depends on the important fact that chills and reactions following intravenous therapy are due to the presence of foreign protein. This protein is either extraneous matter present in the distilled water or the remains of changed blood proteins from a previous intravenous injection. Unless the instruments are cleaned with the greatest care immediately after a transfusion, minute blood clots and other foreign matter remain in the apparatus that may cause a chill in the next case.

The main step, therefore, is the cleansing of instruments and the preparation of solutions so that they are rendered absolutely free of foreign protein. This can be done effectively only when the cleansing of the apparatus and the preparation of the solution used for intravenous therapy are centralized. Only apparatus and solutions properly prepared by competent technicians in the central preparation room should be used for intraveious purposes.

Since adopting this procedure at Mt. Sinai Hospital the authors report a reduction in the number of chills following citrate blood transfusion from 12 per cent to 1 per cent.

Their conclusions are that:

1. Post-transfusion chills are avoidable.

2. Chills are not due to the mixture of the blood with sodium citrate.

3. Elimination of the foreign protein element will reduce the number of chills to a minimum.

4. The use of solutions (sodium citrate and physiologic sodium chloride) prepared with triple distilled water is indispensable.

5. If instruments and solutions are prepared properly, citrate transfusions can be used with safety in every case requiring a blood transfusion.

The authors of this article lay down the following plan for preparation of the solutions and apparatus:

1. Distilled water, sodium citrate and sodium chloride. Triple distilled water, obtained from a special Barnstead still, is used for the preparation of sodium citrate (30 per cent) and sodium chloride (0.85 per cent).

Sodium citrate (30 per cent) may also be bought on the open market in 5 cc. ampules. One should inquire from the manufacturer for sodium citrate prepared with triple distilled water. For actual use, 1 cc. of this concentrated sodium citrate solution is used for every hundred cubic centimeters of blood.

2. Special cleansing of apparatus. After each transfusion, all parts are separated and washed in cold water for the removal of blood. They are then washed in a dilute solution of green soap to which compound solution of cresol has been added to make up about a 1 per cent solution. They are then thoroughly rinsed in tap water.

All parts are then placed in a large pan containing sodium hydroxide (0.1 per cent solution) and boiled for five minutes. They are then transferred to a large pan containing distilled water, to remove the sodium hydroxide. The glassware and rubber tubing are again washed with triple distilled water and are ready to be assembled and sterilized, either in metal boxes or in special bundles, in the autoclave.

The glassware and rubber tubing are boiled separately. The needles are always sharpened before being treated but boiled for only three minutes in sodium hydroxide solution (0.1 per cent).

3. Preparation of apparatus for the autoclave. (a) for the donor: A bundle containing the apparatus for taking the blood from the donor (two pyrex cylinders 500 and 10cc., respectively, one glass rod, two tourniquets, two needles gage 13 and 15, Luer adapter and rubber tubing, one ampule of 30 per cent sodium citrate). (b) for the recipient: A metal box, as shown in the accompanying illustration, devised by Dr. Turner, director of the hospital, containing glassware, rubber tubing and cannulas for the transfusion into the recipient.

Comment: I agree with every statement made in this important article. For the past two years we have had exactly the same experience at St. Anthony Hospital in Oklahoma City, where we have established a central preparation room for all apparatus and solutions for intravenous pur-

poses. This room is in charge of one of the sisters who has done wonderful work. At our staff meeting tonight, (Feb. 20, 1933), she is to give a statistical report of our results. I am not familiar with her statistics, but I am certain from the experience with my own cases that there will be shown marked reduction in the number of reactions following intravenous therapy in the past two years as compared to the number of reactions previous to that time before we had a central preparation room.

—LeRoy Downing Long.

Pulmonary Complications Following Operation on the Stomach or Duodenum. H. K. Gray, M.D. Proceedings of the Staff Meetings of the Mayo Clinic, Volume 8, Number 5, Feb. 1, 1933.

During the first nine months of 1932, there were 422 operations in the Mayo Clinic on the stomach and duodenum. In 43 cases (10 per cent) postoperative pulmonary complications developed and 20 of the patients died. In all but 2 cases, in which partial atelectasis occurred, the process was infectious and inflammatory.

During the same period of time postoperative pulmonary complications following abdominal hysterectomy was 4.6 per cent, following a simple appendectomy 4 per cent and following cholecystectomy 6.9 per cent.

These figures bear out the belief that operation in the lower part of the abdomen is attended with a lower rate of pulmonary complications than with operation in the upper part of the abdomen. No single factor can be held responsible for such complications. It is difficult to accept the opinion that pulmonary complications are usually due to infected emboli from the field of operation because venous communication must be direct. If this were true the pulmonary complications following cholecystectomy, for example, would be accompanied by infectious processes in the liver. In actual experience this seldom occurs.

The hypothesis that these pulmonary complications are often caused by inspiration of infected material during the operation or immediately afterward seems plausible in the stomach cases where there is always a certain amount of manipulation which could cause a reflux of gastric content. With inhibition of the cough reflex in general anesthesia some of the gastric content may return down the trachea and lodge in the lung. However, pulmonary complications occur after spinal anesthesia and after local anesthesia almost as frequently as after general anesthesia. Moreover, spinal anesthesia is not entirely satisfactory for operations on the stomach.

The lymphatic system may be involved in the production of pulmonary complications following operations in the abdomen.

Since the studies of Higgins and Graham in 1928, this possibility has been considered more carefully. The anatomic relations of the lymphatic vessels would seem to make this a more plausible avenue than the venous system.

Operations in the upper part of the abdomen are attended almost invariably by a decrease in the activity of the diaphragm and as a result two factors are brought into play that predispose to pulmonary complications; namely, decreased vital capacity, and sluggish flow in the diaphragmatic lymphatic system.

In view of the fact that there is probably no single factor responsible for pulmonary complications following operation on the stomach and duodenum (and elsewhere in the abdomen), it is suggested that preoperative prophylaxis, operative prophylaxis and postoperative prophylaxis be carried out in the hope of thereby decreasing the incidence of this complication.

Preoperative Prophylaxis: Ascertain the possibility of respiratory infection, however insignificant it may be, and, if possible, postpone the operation. At least a week should intervene between the subsidence of such a condition and any contemplated operative procedure within the abdomen.

Operative Prophylaxis: The operating room should be kept warm, at least 78 degrees Fahrenheit. Inhalation anesthesia, preferably gas, oxygen and ether sequence, has been found to be the most satisfactory. Ether should be used only in sufficient quantities to insure the necessary relaxation. Following operation hyperventilation by means of oxygen and carbon dioxide should be carried out for several minutes before the patient is removed from the operating table. Speed and gentleness are attributes of inestimable value. An indwelling stomach tube during the operation and afterwards offers interesting possibilities in regard to the reduction of pulmonary complications. The adhesive straps on the dressing should be applied diagonally so as to not cause constriction of the costal arch, since this will hamper thoracic respiration. The Trendelenburg position decreases the possibility of aspiration of infectious material. This factor may explain part of the discrepancy in the incidence of postoperative pulmonary complications in operations on the pelvis, as compared with operations on the upper part of the abdomen.

Postoperative Prophylaxis: At the conclusion of the operation, avoid thoughtless exposure of the body. Rubbing the thorax with warm alcohol and immediate application of heated blankets decreases heat loss which undoubtedly contributes to this condition. The patient should be placed in bed in a partial Trendelenburg position until consciousness returns, when he may be placed in a modified Fowler's position. The carbon dioxide oxygen mixture of 10 per cent carbon dioxide and 90 per cent oxygen may be given for one or two minutes every hour for the first six or eight hours immediately following operation.

Comment: The report from this great clinic shows that there are still, even in the best hands, far too many pulmonary complications following upper abddominal operations. The details mentioned are all helpful in preventing pulmonary complications.

—LeRoy Downing Long.

UROLOGY and SYPHILOLOGY

Edited by Dr. S. D. Neely, M.D.
Muskogee, Okla.

The Experimental Production of Urinary Calculi. Charles C. Higgins, Cleveland, Ohio, Journal of Urology for February, 1933, page 157.

The author ran a series of two hundred albino rats on a vitamin A deficiency diet, with 25 controls. Sand was found in the bladder of one con-

trol, no other calculi found in the controls. The first thirty days urinary calculi were infrequent in the rats on vitamin deficiency diet. At the end of two hundred days 88% of these rats showed urinary calculi. Alkalinuria was a constant finding in the rats on this diet. Keratinization of the epithelium of the urologic tract was usually present after ten weeks. Urinary infection developed in a very large per cent of these rats. These factors may be contributing etiologic factors in producing urinary calculi.

The author noted that in one of four rats who had bladder calculi demonstrated by X-ray disappeared after the institution of vitamin A to the diet, for five weeks, another disappeared after seven weeks, and in the third necropsy failed to demonstrate calculi four and one-half weeks after taking vitamin A. The fourth case is at present receiving this diet, sufficient time having not elapsed. The calculi produced in this series of rats varied in size, 0.5 to 6.0 in diameter, usually spherical in shape, light brown color. They contained chiefly calcium phosphate. No oxalates or uric acid was present. Many calculi found at necropsy could not be demonstrated by X-ray.

Upper Urinary Tract and Adjacent Organs—The Differential Diagnosis of Pathological Lesions. William E. Stevens, California and Western Medicine, September, 1932, page 160.

The author states that a complete urological study is indicated in all obscure abdominal conditions. The urologist should have some knowledge of all of the pathologic conditions of the abdomen and pelvis, as an examination of all abdominal and pelvic organs is necessary when the diagnosis of reno-ureteral lesion is uncertain. The diagnosis of chronic appendicitis should be looked upon with suspicion, and operation is not to be considered until a thorough investigation of the right kidney and ureter has been made. Pain in the lower right abdominal quadrant is seldom due to appendicitis in the absence of other symptoms of this condition. The urologist should be proficient in the interpretation of pyelo-ureterograms, as pyeloureterography is of more importance than any other single procedure in the differiential diagnosis of lesions of the upper urinary tract. Complete history, careful physical examination and laboratory study should always accompany this procedure. Cooperation of the general surgeon, internist, gynecologist and urologist is very often indicated in symptoms suggestive of abdominal pathology.

Constitutional Treatment of Ocular Lues. M. L. Greene, Journal Missouri Medical Association, February, 1933, page 64.

The author states that arsenicals in the treatment of optic atrophy and neuritis must be used with great caution. Many cases of latent lues would do better if arsenicals were not used. In acute luetic ocular conditions palliative treatment with mercury by mouth is inadequate, Antiluetic therapy is often of benefit in treating apparently non-luetic conditions.

Transurethral Prostatic Resection. Clinton N. Peters, Maine Medical Journal, February, 1933, page 4.

In conclusion the author states that this proceedure is feasible and practical in many types of bladder neck obstruction. In malignancies it offers the best way of dealing with the obstruction to the urinary flow with the least possible hardship to the patient. It is too recent a procedure to draw conclusions as to end results or its value in replacing prostatectomy in all cases. It is a major surgical operation for the use of trained instrumenteurs accustomed to dealing with bladder neck pathology.

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cedure is feasible and practical in many types of

The Inheritance of Syphilis. R. Matzenaeur, Wein. klin Wchnschr. Vienna, 1932, XLV. 1337.

From his own experience and a discussion of the literature the author concludes that congenital syphilis is transmitted only thru the placenta, and that no proof has been given of inheritance from the father. The author believes that there is no congenital syphilis unless the mother is syphilitic.

Diseases of Common Interest to The Dentist and Oto-Laryngologist. R. T. Atkins, New York State Journal of Medicine, 1932. Vol. 32.

The most common lesions of syphilis that occur in the mouth are the mucous patches of the secondary stage which are often found on the hard palate, lips, gums, and sides of the tongue. The primary lesion may be located on the lips, tongue, palate or tonsil. Tertiary lesions involving the jaws may cause changes in the structure of the mouth.

The Scrotum. C. B. Taylor, Southern Medical Journal, February, 1933, page 187.

The author presents a series of 428 cases of pathology in the scrotum, epididymitis 227, hydrocele, 108, varicocele 38, orchitis 26. Others including eight conditions embraced the remainder. Gumma of the testicle at times offers difficulty in diagnosis, its slow growth, absence of pain, uniformity of outline, and firm consistency make the differential diagnosis from malignancy difficult. A positive serological test is suggestive but not conclusive. Quite often a therapeutic test is necessary. If no response is received in ten to fifteen days on antiluetic treatment surgery should be resorted to. Tumors of the testicle are almost invariably malignant. They are fairly rapid in growth, rarely present a nodular appearance, the mass being smooth, firm and heavy. There is no translucency. The epididymis is not involved and can be outlined. One of the commonest complications of gonorrhea is epididymitis, which presents no difficulty in diagnosis, the history of infection, suddenness of onset, rise in temperature, extensive pain, hardening and tumefaction of epididymis, redness and heat present a picture that is easily read. This author states that acute epididymitis should be operated upon immediately for relief of pain and to facilitate return to industry.

BOOK REVIEWS

Office Surgery, (from Everyday Practice Series, edited by Harlow Brooks, M.D.). By Fenwick Beekman, M.D., Visiting Surgeon, Bellevue Hospital; Visiting Surgeon, Hospital for the Ruptured and Crippled; Consulting Surgeon, Lin-

coln Hospital; Clinical Professor of Surgery, N. Y. University and Bellevue Medical College. Cloth, 94 illustrations. 402 pages. Price $5.00. J. B. Lippincott Company, Philadelphia and London.

In looking through this book, which is a small, practical and very useful edition, we can do no better than to quote from Dr. Brooks' introduction as follows: "A very considerable percentage of the work of the general practitioner is, necessarily, composed of surgery. Only in exceptional instances is the man in general practice, however, called upon to undertake the graver and more complicated surgical operations, though he must meet emergencies on many occasions which demand on his part a very high degree of diagnostic skill, surgical judgment and ability. Unless he is also schooled in what not to do; unless he is fully competent to improve the prospects of his patient, no matter how grave the condition, he will not succeed in general practice.

Unnecessary infection, additional traumatism inflicted in the course of unskilled examinations on the part of an indiscreet practitioner may be the cause of the gravest sort of complications, beyond the skill perhaps, of even the most thoroughly equipped surgeon. Hence every general practitioner must prepare himself at least in the elemental principles of surgery, even though he does not choose to operate himself, except, perhaps, in the most insistent of emergencies."

We have rather continuously heretofore called attention to the role of general practitioners who in most instances see this type of case first and that it is essential that he should early appreciate the dangerous potentialities of even the smallest surgical case. The writer recalls two deaths brought on by merely pulling a hair from the inside of the nose. We would also call attention to this, under fractures "immediate reduction, anaesthesia during reduction and X-ray examination following reduction" If this rule were followed we would greatly reduce the number of bad results and save a great deal of permanent cripplement. With reference to anaesthesia, we too, will remind the reader of the great value of the use of local anaesthesia, directly injected at the site of the fracture. This is a method made popular by Boehler, however, those undertaking this treatment should thoroughly perfect their technic.

The History of Dermatology. By Wm. Allen Pusey, A.M., M.D., LL.D. Professor of Dermatology Emeritus, University of Illinois; Sometime President of the American Dermatological Association and of the American Medical Association. Cloth. Illustrated, 224 pages. 1933. Publisher, Charles C. Thomas, Baltimore, Md., and Springfield, Ill.

Perhaps there is no man in America better informed on all phases of dermatology than Dr. William Allen Pusey. This history of dermatology will naturally appeal to the dermatologists and their allied workers especially. However, it should appeal to all men who are interested in the evolution of medicine. It is worthy of repetition to note that Dr. Pusey says "There is no history of dermatology in English and I have undertaken in this work to supply that lack. It has been necessary, of course, to summarize the subject, but I have recited the story in sufficient fullness to include most of the significant details."

This volume contains chapters on:

Early Ancient Dermatology, Egypt to Greece, 3000 B. C. to 300 B. C.

Graeco-Roman, Arabian and Medieval Dermatology, Rome to the Renaissance, 300 B. C. to 1500 A. D.

Dermatology in Early Modern Europe, 1500 to 1750.

Dermatology Finding Itself, 1750 to 1825.

Threshhold of Modern Dermatology, Clinical Dermatology 1800 to 1850.

Threshhold of Modern Dermatology, Laboratory Dermatology, 1800 to 1850.

Modern Dermatology, First Phase, Continental Europe, 1850 to 1900.

Modern Dermatology, First Phase, Great Britain and the United States, 1850 to 1900.

Modern Dermatology, Present Phase, Since 1890.

Hisorical Index of Dermatology.

INTRAVENOUS STREPTOCOCCIC VACCINE THERAPY IN CHRONIC ARTHRITIS

Macnider Wetherby and B. J. Clawson, Minneapolis (Journal A. M. A., June 4, 1932), report that 80 per cent of 365 chronic arthritic patients treated intravenously with a streptococcic vaccine were clinically improved. The degree of improvemen bore no relation to the degree of reaction following the injection. Improvement was manifested in most cases after five weekly injections. The agglutinating titer of the patients' blood rose during the course of treatment. Improvement as a rule did not take place until the titer had risen to 1:6,400 or more. Treated patients' serums which agglutinated streptococci in dilutions of 1:6,400 or more had a much higher bactericidal power for streptococci than the serums of untreated chronic arthritic patients. The height of the agglutinating titer in most cases seems to be a reliable indicator of the degree of protective immunity to streptococci possessed by the treated patient.

STREPTOCOCCIC MENINGITIS: RECOVERY IN THREE CASES

Although instances of recovery from streptococcic meningitis are exceedingly few, Emanuel Appelbaum, New York (Journal A. M. A., April 9, 1932), reports three cases in which all the patients were treated conservatively and all recovered. On the basis of his observations in these cases and a review of the literature, he concludes that no specific procedure can be formulated for the treatment of streptococcic meningitis. Early spinal drainage, even though moderate, may be regarded as an important factor in the management of these cases. When streptococcus hemolyticus is the causative organism, it seems quite logical to employ antiscarlatinal serum intraspinally. Surgical intervention should, of course, be resorted to for the removal of definite suppurative foci. Other therapeutic measures must, in the light of the present limited knowledge, be regarded as experimental. Finally, one must always bear in mind the possibility of spontaneous recovery from this disease.

END-RESULTS OF SURGERY IN EXOPHTHALMIC GOITER

Howard M. Clute and J. Ross Veal, Boston (Journal A. M. A., Aug. 20, 1932), point out that the immediate advantages of surgical measures in the treatment of exophthalmic goiter have been convincingly demonstrated and that the operation of subtotal thyroidectomy has been made a safe procedure. They summarize the results of surgical treatment in ninety-seven patients whom they have followed consistently for over five years since operation and have checked by personal examination and basal metabolic estimations. In eighty-two patients complete and satisfactory cure was obtained. In seven other patients slight toxicity is still present, entirely controlled by either secondary operation or compound solution of iodine. In three patients of the group myxedema developed; this was entirely controlled by desiccated thyroid. Four of the patients in the group are still toxic. None of the four are in any way incapacitated, and all are working. One of the patients in the group died following a recent secondary operation for recurrent hyperthyroidism and congestive heart failure. It is therefore, the authors' belief that ninety-two, or 94.8 per cent, of these ninety-seven patients are cured of their disease by subtotal thyroidectomy, and that this figure may be assumed to be a fair estimate of the probability of cure of exophthalmic goiter by adequate surgical measures.

WHEN, AS AND IF

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LOWER FAT DIET IN DIABETES

A group of 150 diabetic patients treated by Joseph H. Barach, Pittsburgh (Journal A. M. A., April 9, 1932), with lower fat and higher carbohydrate diet required no more insulin in most cases and less insulin in many. The author emphasizes the fact that the low fat diet is more satisfactory to the patient and complications of the disease are reduced.

PROLAPSE, CYSTOCELE, RECTOCELE AND TRUE VAGINAL HERNIA

James C. Masson, Rochester, Minn. (Journal A. M. A., Oct. 1, 1932), draws attention to the difference between true vaginal hernia with a sac of peritoneum, which is quite rare except as a postoperative condition, and the condition known as gynecologic hernia. As the latter has no peritoneal sac, he speaks of it as a false hernia. The difference in the pathologic picture of a true vaginal hernia and any of the false type is striking. True hernias are spoken of as anterior and posterior, depending on whether the peritoneal sac which is present beneath the vaginal mucous membrane dissects along the anterior or posterior vaginal wall. Under this classification should also be added a much larger group of postoperative hernias, developing after hysterectomy. False hernias of the gynecologic type have no peritoneal sac. When the protrusion is in the anterior wall of the vagina it is called cystocele. When the rectum bulges into the vagina it is a rectocele, and when the uterus itself sinks to a lower level than normal, in the vagina, it is uterine prolapse. A fairly accurate idea of the condition is obtained by grading the hernias 1, 2, 3 or 4 according to their size. The author does not consider the types separately as it is seldom that one type exists without some tendency toward the others. If the type is pronounced (graded 3 or 4), it is easily recognized. In the presence of a large cystocele or rectocele the patient will often have to make pressure on the walls of the vagina in order to empty the bladder or rectum, and in the presence of complete prolapse, or procidentia as it is often called, the uterus will lie outside the vulva, and because of the venous stasis, exposure to air and friction as the result of rubbing on the thighs and clothing, will become edematous and ulcerated. However, carcinoma of the cervix seldom develops in such cases. Early in the development of these hernias, however (size graded 1 or 2), they are often overlooked as the cause of abnormal menstruation, bearing down, uncomfortable feeling in the pelvis and lumbosacral backache. As a rule they develop as the direct result of trauma at childbirth, but occasionally one or the other of the conditions, especially a cystocele, will be found without any tendency toward uterine prolapse, ususally in nulliparous women. In such cases increased intra abdominal pressure and poor development in the anterior muscular part of the pelvic diaphragm probably are the predisposing causes. Urethroceles and diverticula of the urethra are frequently mistaken for cystoceles. The treatment of true anterior and posterior vaginal hernia is surgical. Untreated, the hernia tends to enlarge and the use of palliative measures, such as supports of various types, has proved unsatisfactory. No uniform procedure has been adopted in treatment because true vaginal hernia occurs so seldom, and because coexisting malpositions of the pelvic organs vary so greatly in different cases. However, certain well recognized principles governing the treatment of hernia in general must be adhered to in dealing with this type of hernia. Three measures, which the author discusses in detail, must be accomplished; isolation of the sac, disposal of the sac and repair of the defect at the point of egress of the hernia from the abdomen. The means by which these ends can best be accomplished depend somewhat on the condition and position of the pelvic organs.